“CLINICAL STUDY OF ETIOPATHOGENESIS AND MANAGEMENT OF FACIAL NERVE PALSY”

A DISSERTATION SUBMITTED TO THE TAMILNADU
Dr. MGR MEDICAL UNIVERSITY

CHENNAI

In partial fulfilment of the Regulations

for the award of the Degree of

M.S. (OTO-RHINO-LARYNGOLOGY &HEAD AND NECK SURGERY)
BRANCH-IV

DEPARTMENT OF E.N.T &HEAD AND NECK SURGERY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

MAY 2019

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Date:
Place: Tirunelveli

Dr. S.SURESHKUMAR MS(E.N.T) D.L.O,
Professor and HOD of E.N.T,
Department of E.N.T & HEAD AND NECK SURGERY,
Tirunelveli Medical College,
Tirunelveli

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Date:
Place: Tirunelveli

Dr.S.SURESHKUMAR MS(E.N.T)
D.L.O,
Professor and HOD of E.N.T,
Department of E.N.T & HEAD AND NECK SURGERY,
Tirunelveli Medical College,
Tirunelveli

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Date:
Place: Tirunelveli

Dr.S.M.KANNAN, MS., MCh.,
DEAN
Tirunelveli Medical College,
Tirunelveli

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Date:
Place: Tirunelveli

Dr.S.SARANYA, MBBS.,
Postgraduate in ENT & HEAD AND NECK SURGERY,
Department ENT & HEAD AND NECK SURGERY,
Tirunelveli Medical College,

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Date:
Place: Tirunelveli

Dr.S.SARANYA,MBBS.,
Postgraduate in ENT,
Department of ENT&HEAD AND NECK SURGERY,
Tirunelveli Medical College,

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572733-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO:1042/ ENT/2017

PROTOCOL TITLE: CLINICAL STUDY OF ETIOPATHOGENESIS AND MANAGEMENT OF FACIAL NEI.RVE PALSY
PRINCIPAL INVESTIGATOR: Dr.S.SARANYA, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE STUDENT
DEPARTMENT & INSTITUTION:TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear Dr.S.SARANYA, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on 10.03.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of The Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of The validity
4. An annual status report should be submitted.
5. The TIREC will monitor The study
6. At The time of PI's retirement/leaving the institute, The study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in The original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and The revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, The same should be documented.
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   g. Any deviation/violation/waiver in The protocol must be informed.

STANDS APPROVED UNDER SEAL

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Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

Dr.J.SureshDurai, MD
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

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CONTENTS

ABBREVIATION

ASOM	**:**	**Acute Suppurative otitis media**
CSOM	**:**	**Chronic Suppurative otitis media**
MOE	**:**	**Malignant otitis Externa**
FND	**:**	**Facial Nerve Decompression**

INTRODUCTION

Facial nerve has a tortuous course within temporal bone. It has a long course through a bony canal known as fallopian canal. So it is prone to injury than other nerves in the body. Intra temporal lesions are more common cause of facial paralysis. Facial nerve dysfunction causes physical, functional and psychological impact on the quality of life. So early evaluation and management is needed. Various causes of Facial palsy are Idiopathic, Temporal bone fractures, Infections like otitis media (acute and chronic otitis media, tuberculosis), Ramsay hunt syndrome, Malignant otitis externa, tumors, Congenital, Iatrogenic injuries and Autoimmune disorders like multiple sclerosis.

Facial nerve paralysis are diagnosed by its clinical presentation like facial weakness, loss of taste, decreased tear and salivary secretion. Then otoscopic examination of external auditory canal, tympanic membrane, pure tone audiometry and stapedial reflex are needed. Topo diagnostic test are done to find the site of lesion

Radiological investigations like HRCT to know about intratemporal segment of facial nerve and electro physiological testing in cases of trauma to know about the degree of dysfunction and chance for recovery are indicated in facial nerve palsy patients.

Among the cases of facial palsy some patients improved after conservative management. But some patients need surgical intervention to relieve edema and to remove bony fragment impinging on the nerve.

AIM OF THE STUDY

To study about, etiopathogenesis and management of facial nerve palsy

OBJECTIVES

- To study the various etiopathological factors responsible for facial nerve palsy
- To analyse the outcome of medical and surgical management policy adapted
- To find out if early intervention can reduce the progression of disease

INCLUSION CRITERIA

- All lower motor neuron palsy

EXCLUSION CRITERIA

- Upper motor neuron palsy
- Multiple cranial nerve palsy
- Congenital facial palsy
- Iatrogenic facial paralysis

STUDY AREA

Department of ENT, Tirunelveli Medical College Hospital.

STUDY PERIOD

March 2017 – September 2018.

METHOD OF STUDY

Prospective study

STUDY POPULATION

Average 30 – 50 Patients (40)

Patients attending TVMCH ENT Department.

SCOPE OF STUDY

- The study suggest selection of patients required early and active intervention in order to get good outcome

MATERIALS AND METHODS

- The study population consists of facial palsy due to various etiology like Trauma, CSOM, Herpes, Malignant otitis externa & Malignancy.

- Patients attending or referred to ENT OPD, were included in this study. Detailed history taken and otological examination was done in all patients. Pure tone audiometry, topo diagnostic test, electrophysiological testing and computed tomography of Temporal bone was done in selected individuals according to their etiology. Surgery has done to whom it is indicated and others managed conservatively. They were followed up at 2weeks, 1st month, 3rd month, and 6th month and their improvement is assessed clinically.

FACIAL NERVE ANATOMY

It is a mixed nerve. It consists of 10,000 motor, sensory and parasympathetic fibres

Motor root (7000) arise from facial nucleus in lower pons.

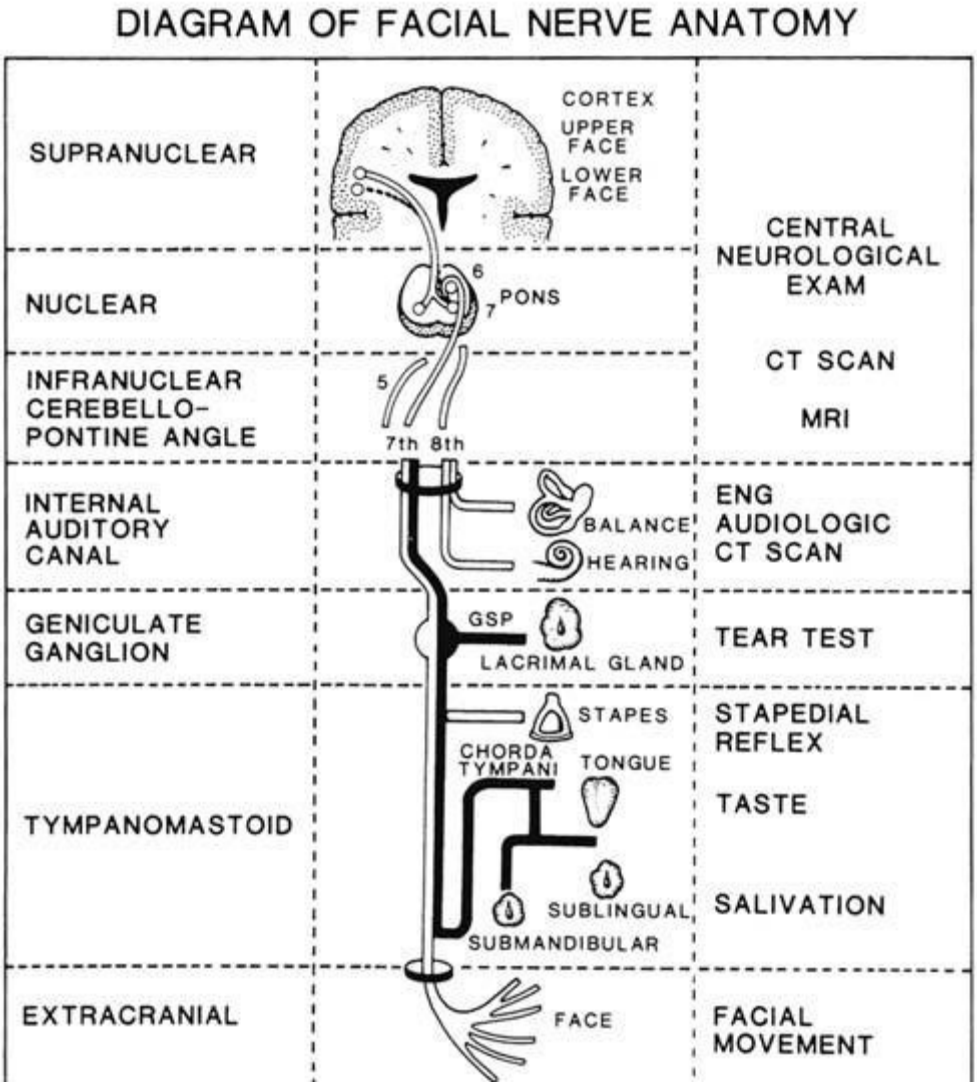
Sensory and parasympathetic fibres (3000) carried by Nervus Intermedius.

It supplies the structures derived from Reichert's cartilage. Facial nerve fibres arises from three nucleus

- Motor nucleus in the caudal pons
- Superior salivatory nucleus- dorsal to the motor nucleus

- Nucleus of tractus solitarius in the medulla oblongata

Upper part of the motor nucleus receives both crossed and uncrossed input from the cortex. But the lower part receives only crossed input.



It has five types of fibres

- General visceral efferent fibres arises from superior salivatory nucleus and they supply submandibular, sublingual and lacrimal glands.
- Special visceral efferent supply muscles of facial expression, stylohyoid, stapedius and posterior belly of digastric
- Special visceral afferent fibres carries taste sensation from anterior two third of tongue.
- Somatic afferent fibres give innervations to the skin of posterosuperior part of external auditory canal.

COURSE OF FACIAL NERVE

Divided into

- Intra cranial part
- Intra temporal part
- Extra temporal part

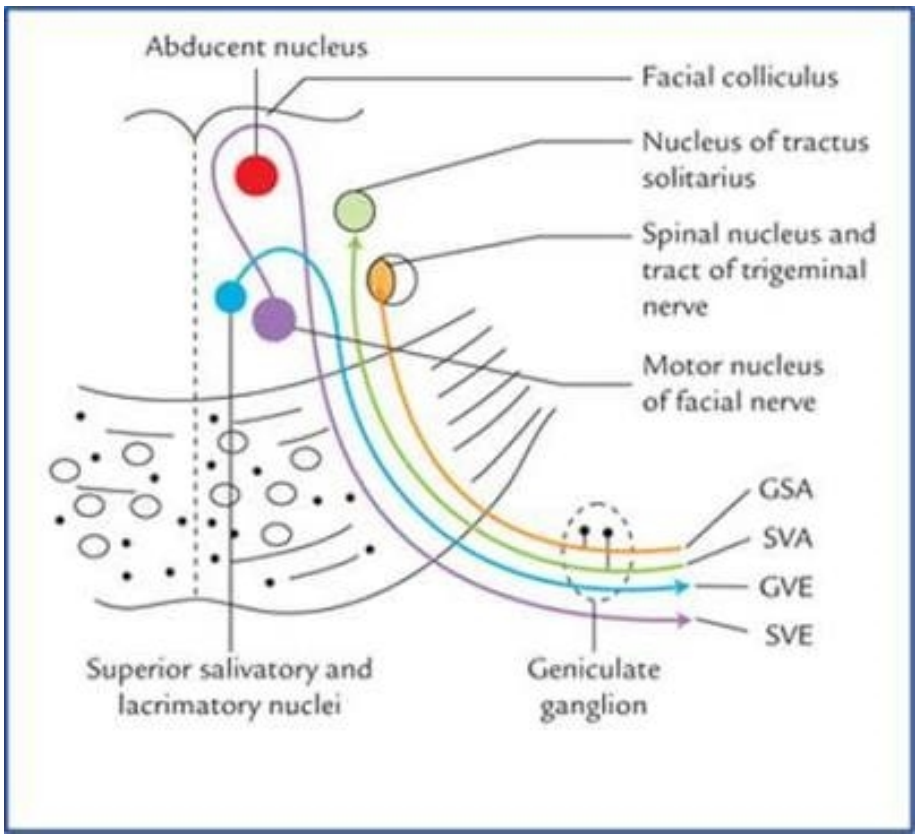
1.Intra cranial part

From brainstem to fundus of internal auditory meatus

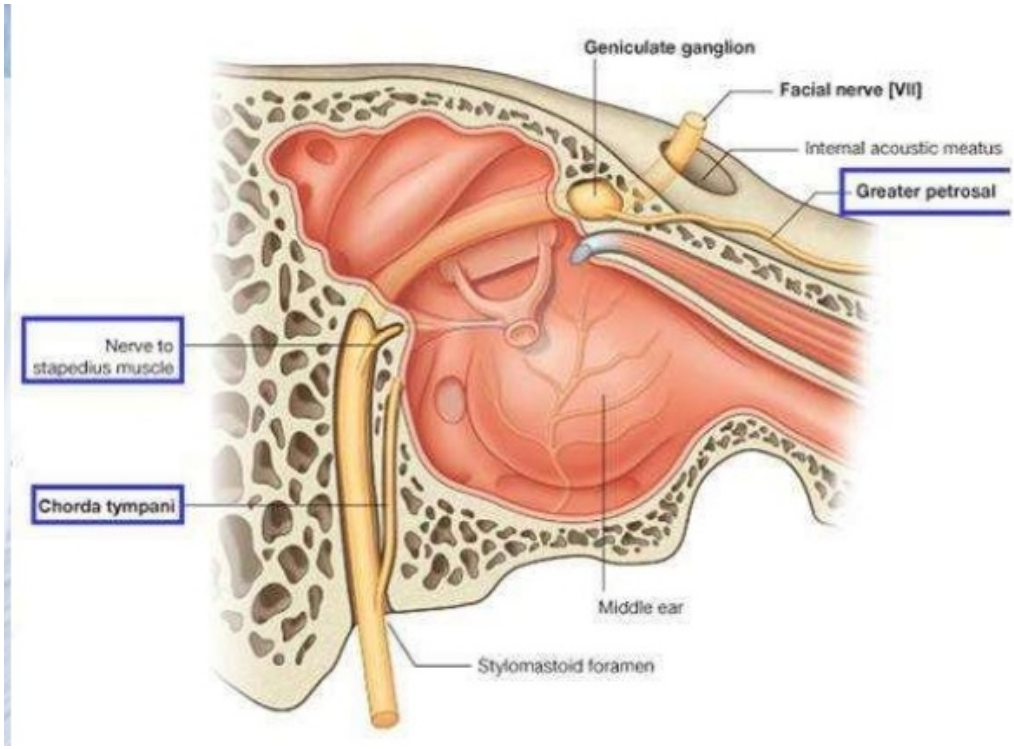
Length 24mm

Facial nerve arise from motor nucleus in the pons and hooks around the sixth nerve nucleus and exists at the pontomedullary junction between olive and restiform bodies. It traverses the cerebellopontine angle along with eight nerve and nerve of wrisberg.

In this segment no perineurium. Only thin layer of piamater covering the nerve. So the nerve is vulnerable to injury at cerebellopontine angle. After enter into internal auditory meatus the nerve runs for 5-12 mm to the fundus of IAM.



2.Intratemporalpart



Has three segments

- Labyrinthine part

  From fundus of IAM to geniculate ganglion.

  Length 4mm.

  Shortest and thinnest segment

  At fundus nerve enters into fallopian canal.

Narrowest part is at its entrance into the canal and its thickness is 0.68mm.

In case of edema or inflammation the nerve get easily compressed.

- Tympanic or horizontal part

  From geniculate ganglion to just above the pyramidal eminence. The nerve takes 75 degree turn posteriorly at the geniculate ganglion. It traverses medial wall of the middle ear below the lateral semicircular canal and above the oval window. It's proximal end lies superior and medial to the cochleariform process and Greater Superficial Petrosal Nerve arises from geniculate ganglion.

Length 8 -11mm

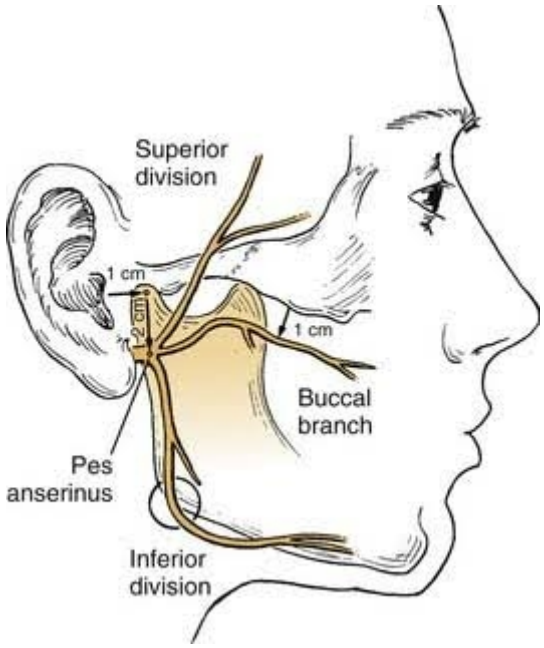
- Mastoid segment

Runs from pyramidal eminence or 2nd genu upto stylomastoid foramen. This segment forms an angle of 95 to 125 degrees with the tympanic portion.it makes an obtuse angle just before enter into stylomastoid foramen.

Length 9 to 16mm

3.Extratemporal part

Starts from stylomastoid foramen to termination of its peripheral branches.



ANATOMICAL VARIATIONS OF INTRATEMPORAL COURSE

- Tympanic segment of the facial nerve runs anterior and inferior to the oval window.
- Vertical segment of the facial nerve runs more posterior and lateral than usual as it runs below the prominence of lateral semicircular canal.
- Mastoid segment of the facial nerve may be bipartite or tripartite.

- Tympanic segment of the nerve over the oval window may have dehiscence. Rarely facial nerve may protrude through this gap and presents as middle ear mass.
- Subarachnoid space of the nerve may extend upto tympanic segment and may spontaneously fistulize into the middle ear and produce csf otorrhoea.

ANATOMIC VARIATIONS OF EXTRATEMPORAL COURSE

KATZ AND CATALANO classified the branching pattern of facial nerve within parotid into five types

- Type1 (25%)

  a. splitting and reunion of zygomatic branch

  b. splitting and reunion of the mandibular branch
- TYPE 2 (14%)

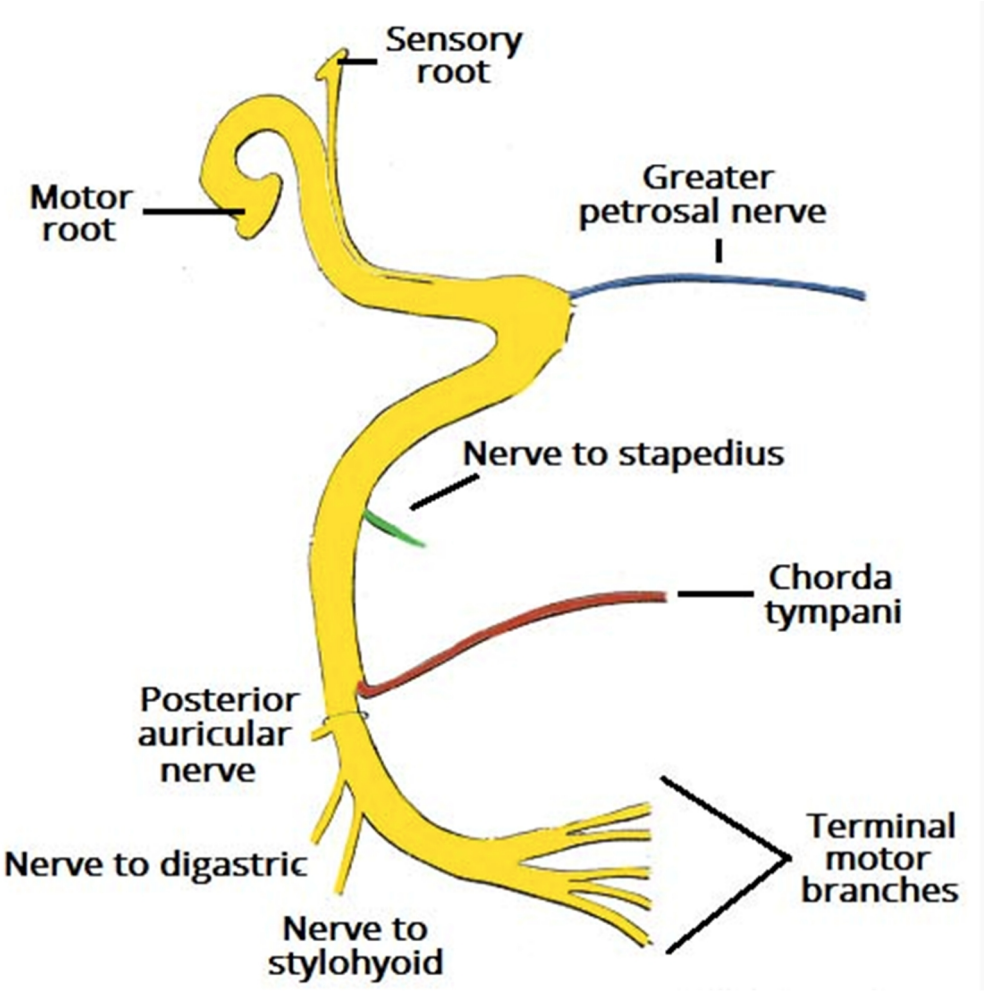
  Buccal branch fuses distally with the zygomatic branch
- TYPE 3 (44%)

  Major communication between buccal branch and other branches
- TYPE 4 (14%)

  Anastomotic branching patterns between major divisions
- TYPE 5 (3%)

  The nerve leaves the skull as more than one trunk

BRANCHES



- Greater superficial petrosal nerve

  Originate from geniculate ganglion and enter into middle cranial fossa through facial hiatus and runs towards foramen lacerum. It carries secretomotor fibres to the lacrimal gland.

- Nerve to stapedius

  Arise from vertical part of nerve near pyramidal eminence.it supplies stapedius muscle

- Chorda tympani nerve

It Joins 4mm proximal to the stylomastoid foramen. It enters into the tympanic cavity through iter chordae posterius and runs between incus and malleus and leaves the cavity by passing through iter chordae anterius (canal of Hugier). It joins with lingual nerve and supplies anterior two third of tongue. It gives secretomotor fibres to submandibular gland

- Sensory fibres join with auricular branch of vagus supply the skin of external auditiory canal
- Posterior auricular branch arises 1-2 mm below stylomastoid foramen. It winds round digatric muscle and extends posteriorly up to anterior surface of mastoid. It divides into auricular and occipital branches
- Muscular branches to stylohyoid and posterior belly of digastric
- Peripheral branches

Within parotid gland it gives two branches

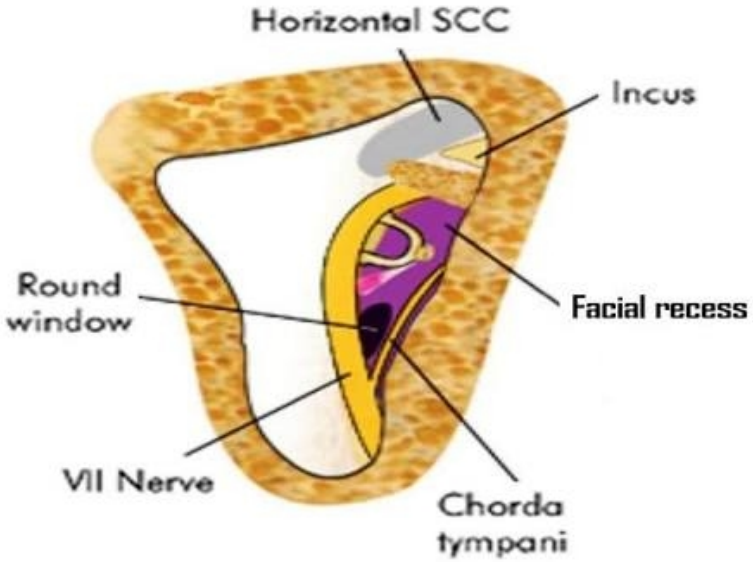
Upper temporofacial

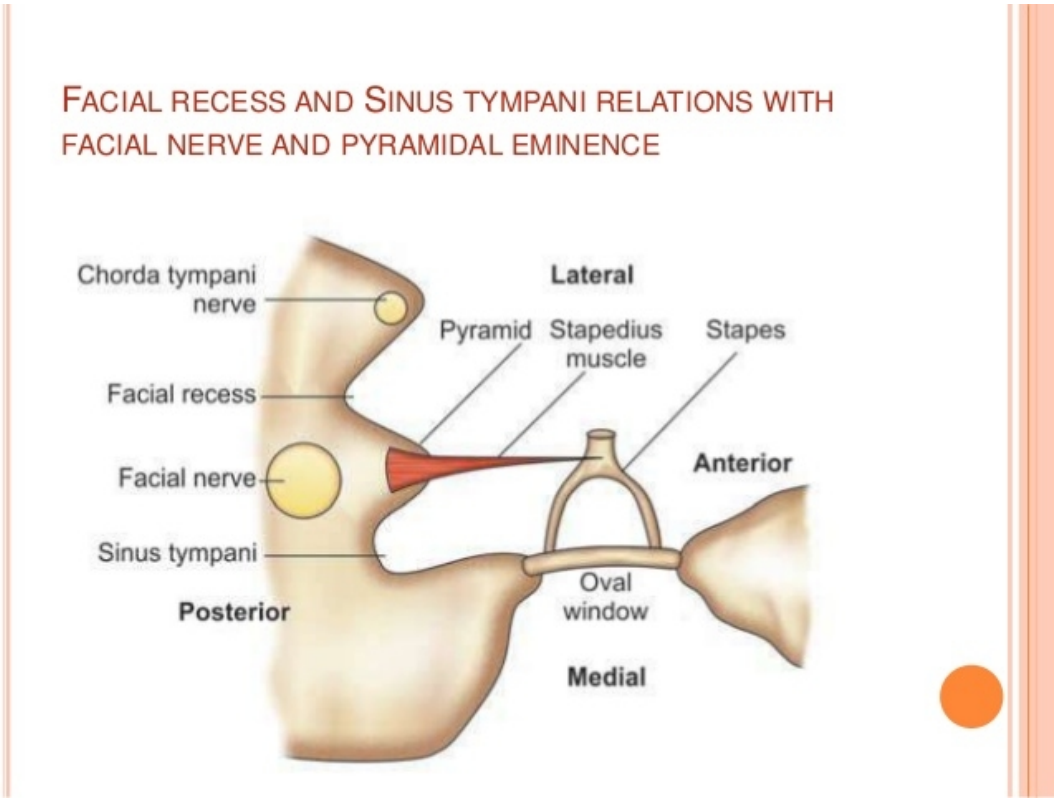
Lower cervicofacial

They further divide into temporal, zygomatic, buccal, marginal mandibular and cervical. Their appearance called pesanserinus

FACIAL RECESS

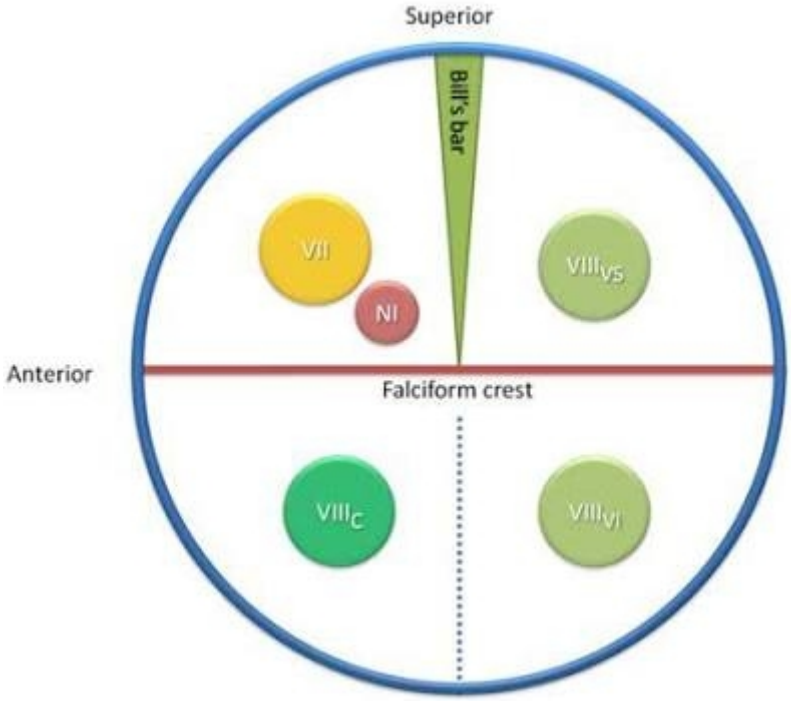
It is bounded superiorly by fossa incudis, medially by vertical segment of the facial nerve and laterally by chorda tympani nerve. In the intact canal wall mastoidectomy it is used to access the middle ear.





INTERNAL AUDITORY CANAL

It measures 8mm in length and 3.4mm in diameter. The medial part of the canal is porus that opens in the posterior cranial fossa. The lateral part of the canal is fundus that abuts the bony labyrinth. Crista falciformis is a horizontal crest divides the canal fundus into superior and inferior portions. Bill's bar is a vertical crest in superior aspect that divides it into anterior and posterior parts. Facial nerve lies anterosuperiorly and superior vestibular nerve lies posterosuperiorly. Inferior vestibular nerve and cochlear nerve lies below the horizontal crest. Bill's bar is an important surgical landmark in translabyrinthine surgery of cerebellopontine angle.



BLOOD SUPPLY

- Stylomastoid artery
- Petrosal branch of middle meningeal artery
- Antero-inferior cerebellar artery.

These three branches anastomoses and forming extrinsic vascular plexus within the epineurium. Intrinsic vascular plexus arises from extrinsic system. This system support the nerve when extrinsic system is disrupted or when the nerve is mobilised from the fallopian canal. Veins accompany the arteries in the facial canal. Lymphatic vessels are located in the epineurium.

SURGICAL LANDMARK

LAND MARKS IN THE MASTOID AND MIDDLE EAR

- COG that is bony ridge hangs from tegmen useful to identify the first genu
- Processus cochleariformis

  Geniculate ganglion (proximal end of tympanic segment) lies just above and medial to it.
- Oval window

  Horizontal part of the nerve lies above it.
- Lateral semicircular canal

Second genu of facial nerve hugs the lower aspect of horizontal canal. This is very constant landmark

- Pyramidal eminence

  Facial nerve lies lateral and posterior to it.

- Short process of incus

  Nerve lies medial to it.

- Digastric ridge

  A line drawn between anterior end of digastric ridge and tip of short process of incus makes facial nerve mastoid segment's course

LANDMARKS FOR EXTRATEMPORAL PART

- Tympanomastoid fissure

  Facial nerve lies 6 to 8 mm deep to this fissure.

- Tragal pointer

  The nerve lies 1cm medial and inferior to the tragus.

- Styloid process

  The nerve lies below tympanic plate and lateral to the base of styloid process.

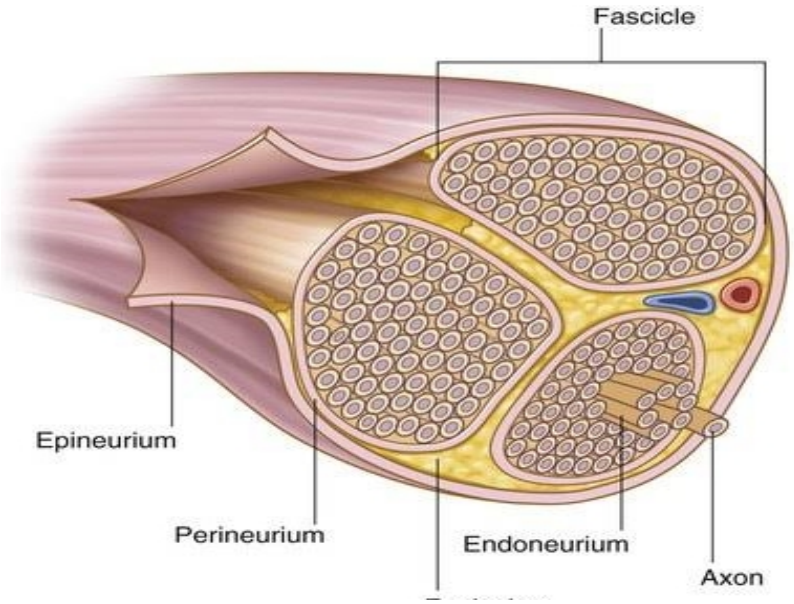
- Posterior belly of digastric

  If this muscle traced backwards towards it's insertion to digastric groove the nerve lies between it and styloid process

- By tracing terminal branches of the nerve backwards

HISTOLOGY OF NERVE

The nerve fibre composed of cell body and axon and is surrounded by layer of myelin secreted by Schwann cells. Each nerve fibre is surrounded by many Schwann cells that provide metabolic support. Each nerve fibre is surrounded by endoneurium forming a tubule. Multiple tubules are bound together in bundles and are covered by perineurium forming fascicles. Multiple fascicles bound together and covered by epineurium.



PATHOLOGY

SEDDON'S CLASSIFICATION (1943)

I. NEUROPRAXIA

Reversible block in the transmission of nerve impulse and is due to pressure on the nerve fibre.

II. AXONOTMESIS

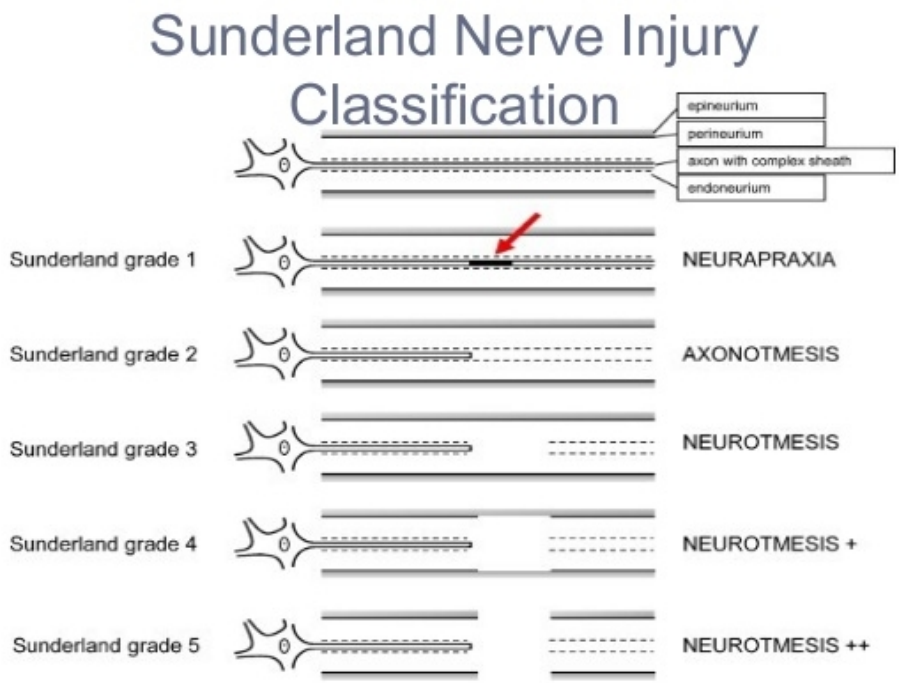
There is blockage of axoplasmic flow. Endoneural tube is intact. But distal wallerian degeneration can occur.

III. NEUROTMESIS

The nerve is totally transected. Wallerian degeneration occurs 3 to 4 days following injury.

SUNDERLAND CLASSIFICATION

It is only for traumatic injuries of the peripheral nerve. It is not for viral, inflammatory or infiltrative lesions.



I. FIRST DEGREE INJURY

Blockage of nerve impulse transmission. Recovery begins 1-4 weeks.complete recovery.

II. SECOND DEGREE INJURY

Here loss of axons. Endoneurium intact. Recovery begins 1-2 months. wallerian degeneration occur.

recovery is usually complete.

III. THIRD DEGREE INJURY

Endoneurium disrupted. Recovery starts 2-4 months.

Incopmplete recovery. Usually complicated by functional sequeale

Second and third degree injuries equal to axonotmesis.

IV. FOURTH DEGREE INJURY

Perineurium disrupted in addition to 3rd degree injury. Partial transaction of nerve occur. Recovery starts 4-18 months. Recovery poor.

V. FIFTH DEGREE INJURY

Epineurium also disrupted in addition to 4th degree injury. Total transaction of nerve occur. Without surgical intervention recovery is not possible.

In third degree injuries or above the regenerating axons enter into endoneurial tube of another axons that result in inappropriate muscle innervations. That produces synkinesis.

The rate of axonal Regeneration is 1mm/day. But rate of regeneration and overall recovery depends upon etiology and severity of injury, degenerative process. patient's age, blood supply, comorbid conditions and concurrent wound infection also influence the recovery.

GRADING OF FACIAL NERVE PARALYSIS

(HOUSE –BRACKMANN GRADING)

DEGREE OF INJURY	GRADE	DEFINITION
Normal(1st degree)	One	Normal symmetrical function
Mild dysfunction(1-2 degree)	Two	Slight weakness on close inspection. Complete eye closure. Slight asymmetry of smile.
Moderate dysfunction (2-3 degree)	Three	Obvious not disfiguring weakness. Complete eye closure with maximum effort. Asymmetric mouth movement. Inability to lift eyebrow. Obvious synkinesis.
Moderately severe dysfunction(3rd degree)	Four	Obvious disfiguring weakness. Incomplete eye closure with maximum effort. Asymmetric mouth movement. Inability to lift eye brow. Severe synkinesis.
Severe dysfunction(3-4th degree)	Five	Incomplete eye closure Slight movement of angle of mouth. Synkinesis absent.
Total paralysis	Six	No movement and loss of tone Synkinesis absent.

Another grading system known as Repaired Facial Nerve Recovery scale(RFNRS). Here we are using letters instead of numbers.

REPAIRED FACIAL NERVE RECOVERY SCALE

SCORE	FUNCTION
A	Normal facial function
B	Slight movement of forehead, independent movement of eyelids and mouth, slight mass motion
C	Strong closure of eyelids, some mass motion, no forehead movement
D	Incomplete eyelid closure, significant mass motion, good tone
E	Poor tone and minimal movement in any branch
F	No movement

EVALUATION

Factors to be assessed are date of onset and progression, duration of symptoms, risk factors and comorbid conditions.

PHYSICAL EXAMINATION

- Auricle and external ear examination
- Otoscopic or microscopic evaluation of tympanic membrane
- Complete head and neck examination
- All branches of facial nerve should be examined
- Cerebellar and other cranial nerve examination

INVESTIGATIONS

- Blood investigations(CBC,ESR,SUGAR)
- Serological test for syphilis and HIV
- Pure tone audiometry,
- In case of retero cochlear lesions, acoustic reflex decay or auditory brain stem response

SPECIAL INVESTIGATIONS

To know about the cause of palsy, site of lesion and the prognosis

TOPODIAGNOSTIC TEST

To locate the level of lesion

TEST	THE NERVE TO BE TESTED	OUTCOME
Schirmer's test	Greater superficial petrosal nerve	>75% decrease in one side or both side decrease in less than 10 mm at 5 minutes.
Stapedial reflex	Nerve to stapedius	Present /absent
Electrogustometry	Chorda tympani nerve	Taste threshold is compared between both sides
Salivary flow testing	Chorda tympani nerve	Salivary flow less than 25% is abnormal

ELECTRODIAGNOSTIC TEST

Used to assess degree of dysfunction of the neve in the presence of viable neuropraxic axons and also to predict potential of recovery.

MINIMUM NERVE EXCITABILITY TEST

Comparing current threshold needed to produce minimal contraction of muscle on both sides

Difference of >3.5mA is significant. That is indicative of poor recovery.

MAXIMUM STIMULATION TEST

Contraction of muscle at maximum stimulation of nerve is compared between two sides. Response may be equal, reduced or absent.

Incomplete recovery is associated with loss of response within ten days.

ELECTRONEURONOGRAPHY

Facial nerve is stimulated where it exists the temporal bone

(stylomastoid foramen) and the evoked compound muscle action potential is recorded by using surface electrode. We are calculating the percentage of degenerated fibres. The reduction in the CAMP correlates with histological axonal loss.

This test is not useful upto the fourth day of injury. Because it takes about three days for the wallerian degeneration to complete. It is also not useful after three weeks because of desynchronization of nerve fibres.

It's mostly indicated in acute onset complete paralysis. In traumatic injury surgery should be done within three weeks if >90% of degeneration occur within six days of injury.

In Bell's palsy >90% degeneration developed within 14 days of complete paralysis results in poor recovery. It is not useful in Ramsay Hunt Syndrome where there is multiple sites of involvement.

ELECTROMYOGRAPHY

It records active motor unit potentials of orbicularis oris and oculi both during rest and voluntary contraction. Fibrillation potentials due to wallerian degeneration occur within two to three weeks of injury. Polyphasic potentials due to reinnervation precedes the recovery by 6-12 weeks. It is most useful to make decision regarding surgery in case of long standing paralysis.

Fibrillation potentials indicate viable motor end plate. Surgery is indicated to maintain nerve continuity. Polyphasic potentials indicate regeneration so surgery is not indicated.

LIMITATIONS OF ELECTROPHYSIOLOGICAL TEST

- It is not useful in incomplete paralysis
- It is not useful in immediate post paralysis
- It stimulates normal and neuropraxic fibres. it could not differentiate whether the fibers are in axonotmesis or neurotmesis.

IMAGING

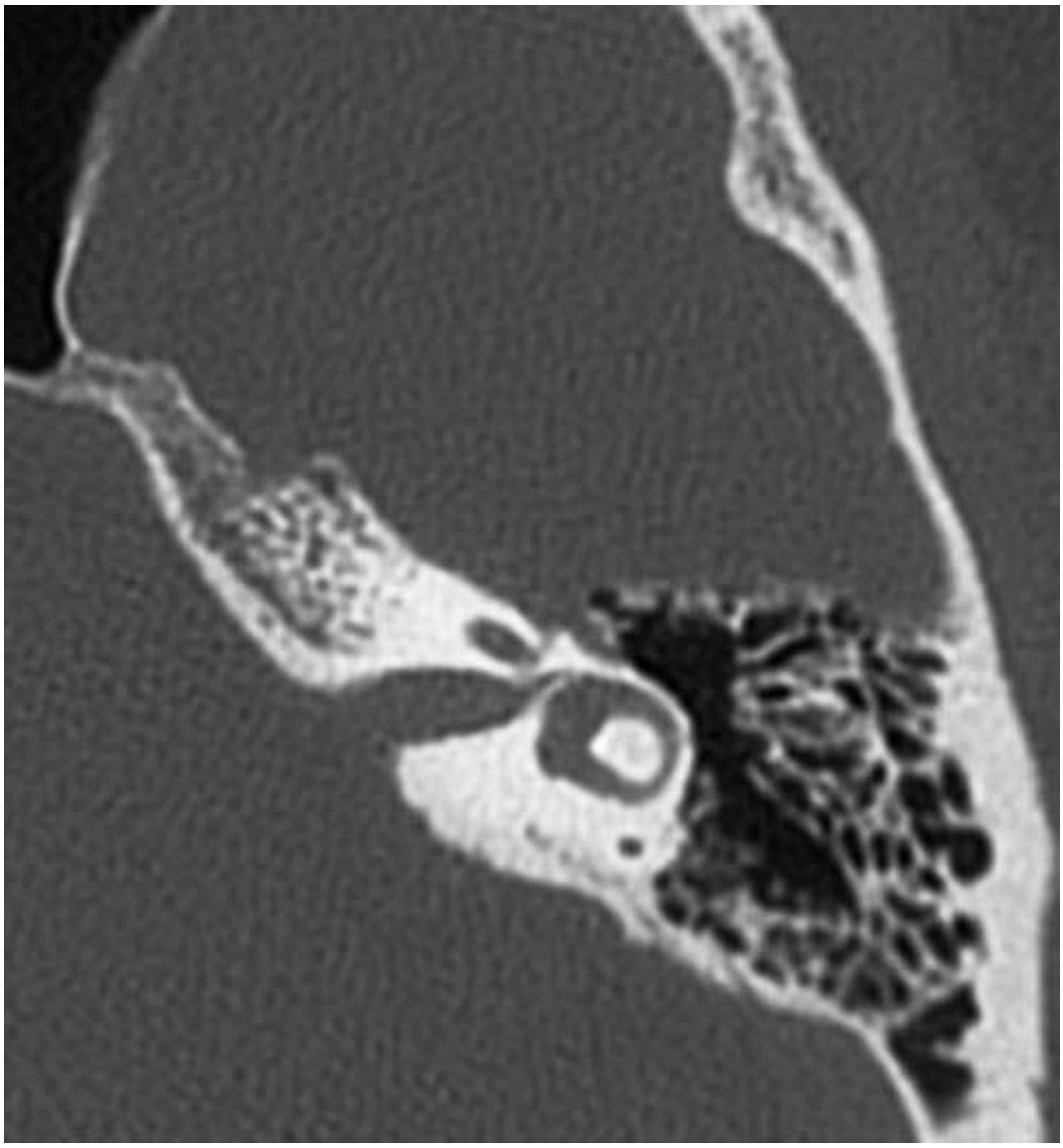
- HRCT

In CT tympanic portion of facial nerve identified at the level of incus body and its short process in axial cuts. Then the nerve identified proximally towards labyrinthine portion and distally towards vertical segment. Labyrinthine part seen as banana shaped in axial cuts. It is seen as medial part of two circular eyes above the cochlea in coronal cuts.

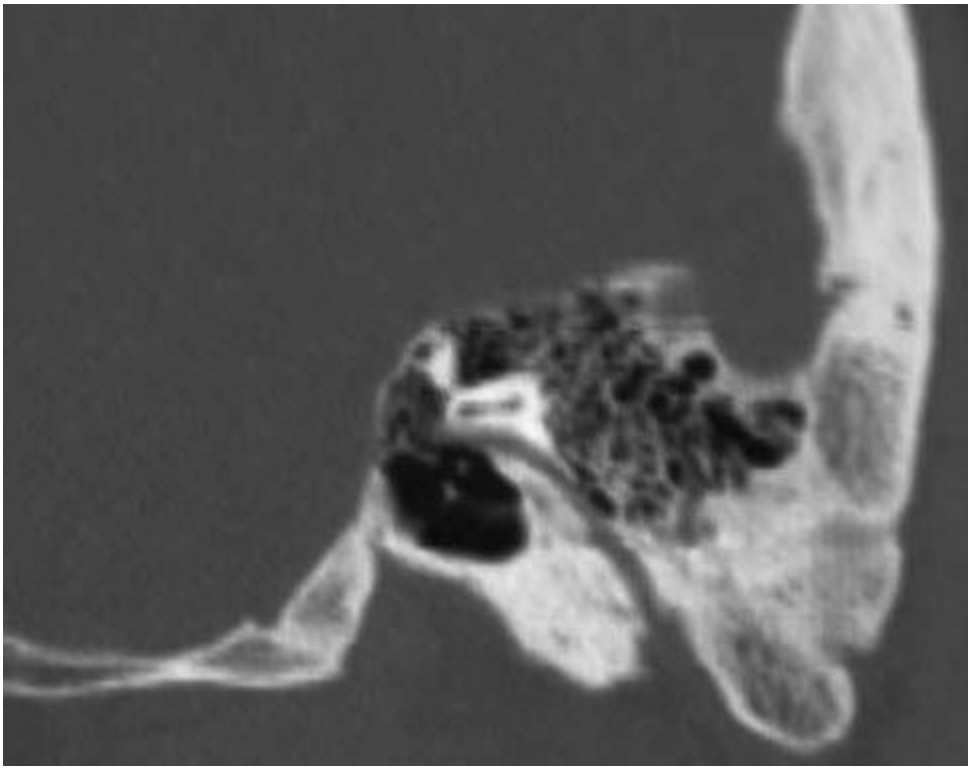
Sulcus for geniculate ganglion can be seen in coronal cuts. In coronal cuts vertical part of facial nerve can be easily followed from stylomastoid foramen towards proximally. Other way is locate pyramid or stapedius muscle. That part of nerve lies behind it.

Tangential line from basal turn of cochlea falls within 1 mm of facial nerve that is a B line described by Bance and Erb. Line connected from stylomastoid foramen to a point lateral to reteromandibular vein also indicates plane of facial nerve.

By using CT we can visualize fallopian canal in case trauma, CSOM and malignancies



CT Scan showing labyrinthine segment of the nerve

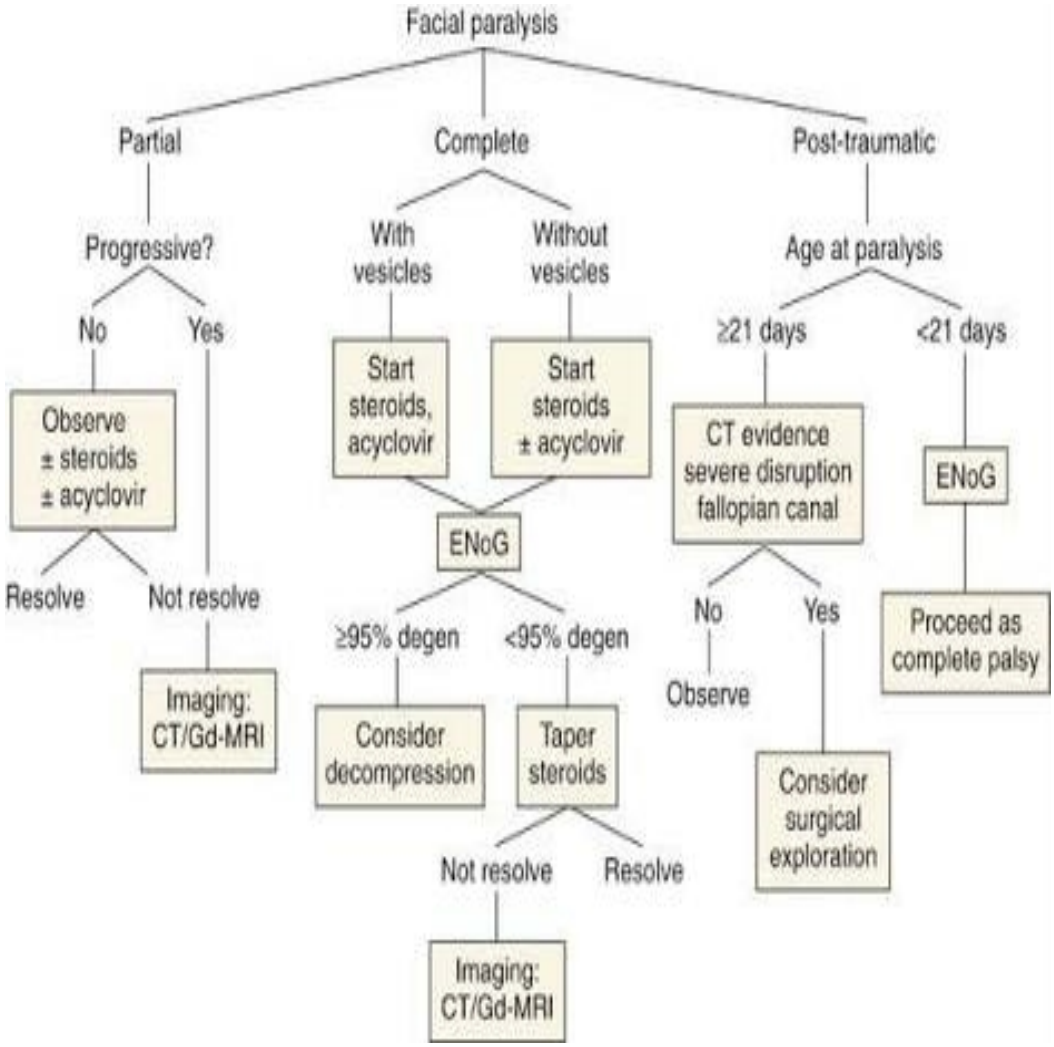


Obligue sagital view showing mastoid segment of the nerve

- MRI

Because of rich arteriovenous plexus around the facial nerve we can observe the enhancement on T1 weighted images with use of contrast materials.

Gives better idea about soft tissues in case of inflammation and Neoplasms**.**



Sequale of degeneration of the nerve

- Facial asymmetry
- Synkinesis

  Smiling associated with involuntary eye closure

  Sweating over the preauricular area during chewing known as FREY'S SYNDROME

- Lacrimation while eating known as CROCODILE TEARS
- Clonic facial spasm (tics)

CAUSES OF FACIAL PALSY

TRAUMA

- Skull base fracture
- Penetrating injuries to middle ear
- Facial injuries
- Barotraumas

INFECTIONS

- Otitis externa
- Otitis media

  ASOM

  CSOM/ CP with granulations

  CSOM/AAD with cholesteatoma
- Mastoiditis
- Ramsay hunt syndrome
- Poliomyelitis
- Mumps
- Infectious mononucleosis
- Tuberculosis
- Syphilis
- Mucormycosis
- Lyme disease

METABOLIC

- Diabetes mellitus
- Hypertension
- Hyperthyroidism
- Pregnancy

TOXIC

- Thalidomide
- Tetanus
- Diphtheria

IATROGENIC

- Mastoid surgery
- Parotid surgery

NEOPLASTIC

- Glomus jugulare
- Facial nerve tumor
- schwannoma
- Carcinoma
- Menigioma

IDIOPATHIC

- Bell's
- Melkersson Rosenthal syndrome
- Autoimmune syndrome
- Temporal arteritis
- Sarcoidosis
- Gullian Barre syndrome

BIRTH

- Moulding
- Forceps delivery
- Moebius syndrome
- Dystropia myotonia

NEUROLOGICAL

- Millard –Gubler syndrome

BELL'S PALSY

Incidence 20 to 30 cases per 1,00,000 individuals per year

TAVERNER DIAGNOSTIC CRITERIA

- Paralysis or paresis of muscle groups on one side of face
- Sudden onset
- Absence of signs of central nervous system diseases
- Absence of signs of ear or CPA diseases

ETIOLOGY

Most accepted is viral etiology. The cause of paralysis is viral neuropathy alone or ischemic neuropathy secondary to viral infection. It is due to reactivation of varicella zoster virus in the geniculate ganglion that is initiated by various stresses such as heterotropicviruses(viruses other than herpes), physical or metabolic stresses. It is a part of cranial nerve polyneuritis.Among which facial palsy is common finding.

Majority are recovered completely. Poor prognosis seen in old age, complete paralysis at onset, incomplete palsy with late onset recovery.

Recovery is usually within 3 months in two third of patients with bell's palsy. More than 6 months recovery could not be expected

Recurrence is 7-12%.

TREATMENT

Prednisolone 1mg/kg/day for five days followed by tapering dose for ten days and ten day course of oral acyclovir 200-400 mg five times daily.

MARSH AND COKER CRITERIA of surgical indications

- Complete denervation
- Incomplete return of function in 60 days
- Paralysis for more than 4 to 6 weeks
- Recurrent facial palsy
- Nerve excitability shows difference of 3.5mA on both the sides

FACIAL NERVE TRAUMA

- Temporal bone trauma
- Maxillo facial trauma
- Gun shot injuries

TEMPORAL BONE TRAUMA

Divided into longitudinal, transverse and mixed fractures.

Facial paralysis due to

- Complete or incomplete transection of the nerve
- Bony fragments compressing the nerve
- Edema of the nerve as a part of inflammation due to trauma

LONGITUDINAL FRACTURES

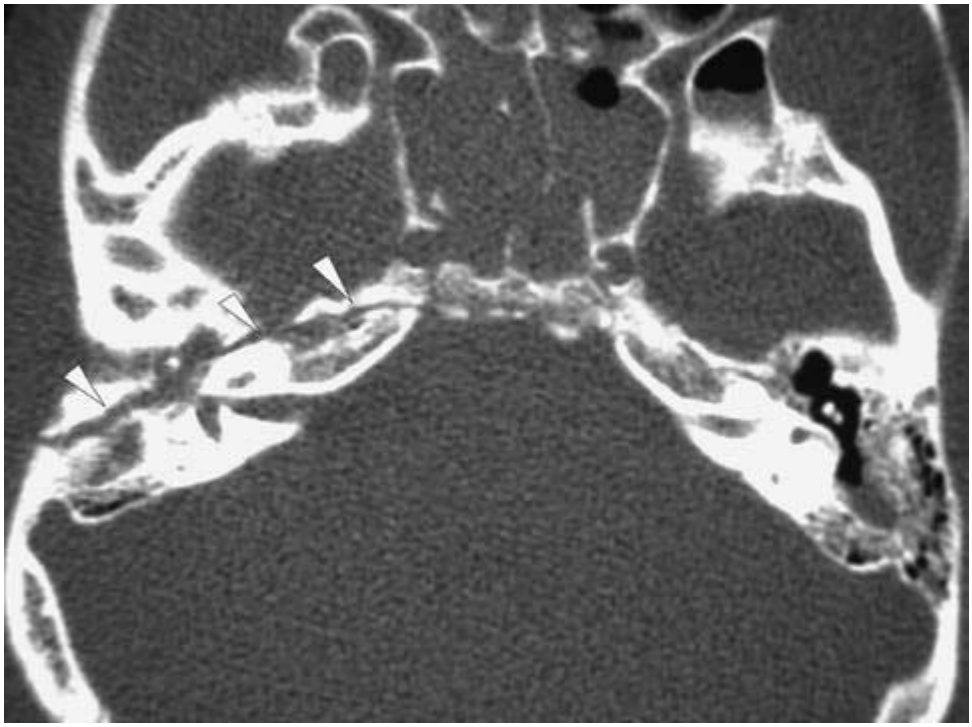
- Most common.
- incidence 70-90%.
- Caused by blow to temporal or parietal areas
- Fracture line runs along the petrous part of the bone.
- Starts in squamous part and extends in EAC posterosuperiorly and roof of the middle ear and anterior to the labyrinth and ends in MCF.
- Involve middle ear and ossicles, occasionally facial nerve canal and inner ear spared.

SYMPTOMS AND SIGNS

Bleeding into the ear (Hemotympanum) and TM perforation

Ossicular disruption leading into conductive hearing loss and some have sensorineural hearing loss due to concussive damage

- Facial paralysis occur in 20-25% of cases. Common site distal to geniculate ganglion.
- Onset of palsy is immediate or delayed and partial or complete.
- Good prognosis.



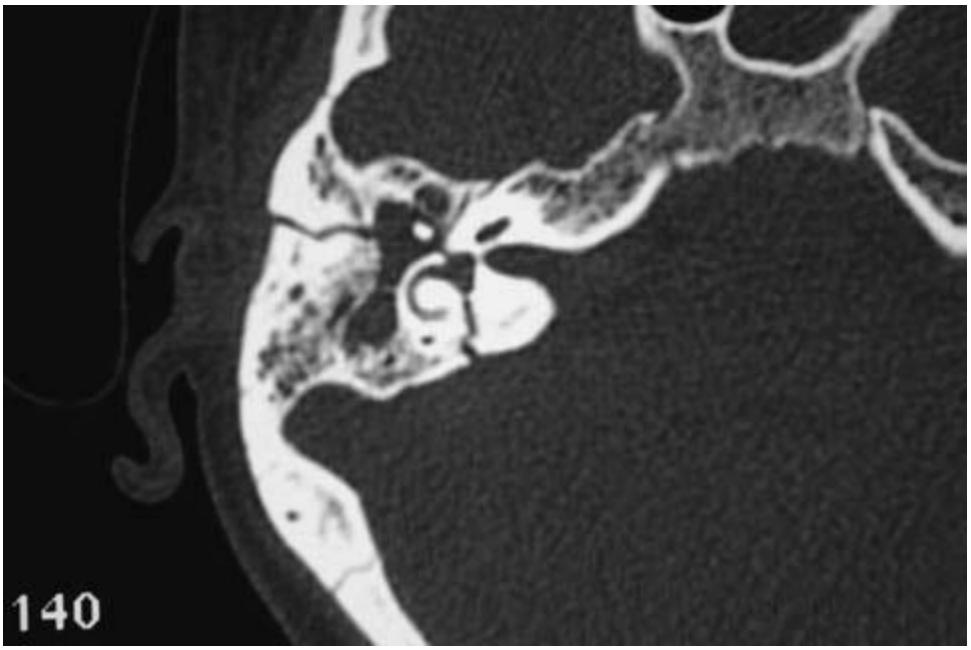
TRANSVERSE FRACTURE

- Less common.
- Incidence 10 -30%
- Caused by frontal or occipital blow
- Fracture line runs perpendicular to the long axis of petrous part of pyramid.
- fracture line starts in MCF crosses the petrous pyramid and end at foramen magnum.
- Inner ear involved

SYMPTOMS AND SIGNS

- Sensorineural hearing loss
- Vertigo and nystagmus
- Facial nerve paralysis. Incidene 50%.Common site is proximal to the geniculate ganglion and anywhere from IAC.
- Onset of palsy usually immediate and complete.

  Temporal bone fractures heals in a fibrous way. So it leaves a fissure for rest of life.



MANAGEMENT

Delayed palsy or acute incomplete palsy without progression prognosis was excellent. Surgical exploration is not required. In acute complete palsy if ENOG shows >90%degeneration within six days of injury surgical exploration is required.

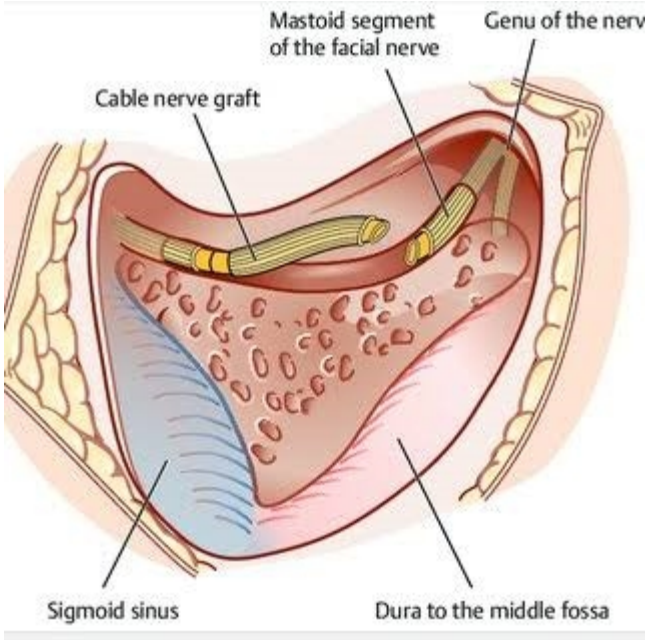
Surgery is needed to

- to prevent ischemic injury
- Remove bony fragments that penetrating the nerve.
- In case of transection, re-establish the nerve continuity.

In case of delayed post injury facial nerve palsy, decision for surgery depends on CT findings or EMG RESULTS. Surgical exploration is indicted if return of facial function is not observed within 6-12months after injury or In EMG no signs of polyphasic potentials suggestive of reinnervation.

Surgery is indicated to achieve nerve continuity by end to end anastomosis or interposition grafting or rerouting. If EMG shows long term denervation static or dynamic facial reanimation procedure is required.

IATROGENIC FACIAL PALSY



Incidence between 0.6 and 3.6%.

Thermal facial nerve injury can occur during drilling with diamond burr. Removal of diseases near fallopian canal can alter the vascularity of the nerve leads to the ischemia with temporary facial palsy. In middle ear surgery most common site is tympanic segment because of dehiscence. In mastoid surgery most common site is second genu.

FACIAL PALSY IN INTACT CANAL WALL MASTOIDECTOMY

- Inadvertent handling of incus can injure second genu if dehiscent.
- Widening of aditus from antrum dehiscent second genu can injured.
- Graft material for ossicular reconstruction may injure dehiscent horizontal part and second genu.
- While performing posterior tympanotomy mastoid part may injured.
- In case of low lying dura and contracted antrum if drill more anteriorly and inferiorly mastoid part may be injured.

IN CANAL WALL DOWN MASTOIDECTOMY

- While lowering facial ridge if nerve is dehiscent
- While operating in floor of fossa incudes or removal of posterior buttress.
- In infants if post auricular incision extended inferiorly

If injury is recognised intra operatively, exploration with decompression should be undertaken.

If facial paralysis observed immediately after surgery and the nerve was identified intra operatively, few hours of observation is needed for local anesthesia induced weakness to resolve. Mastoid dressing and canal packing if present should be removed during the period of observation.

If paralysis is incomplete oral steroids should be started. In case of complete palsy exploration is indicated if >90%degeneration in ENOG.

In case of delayed palsy etiology may be reactivation of VZV or HSV in geniculate ganglion.

Delayed palsy usually occur between third and twelfth post injury day. prednisolone and acyclovir should be started.

The nerve can also get injured in parotid surgery and cerebello pontine tumor surgery. In neonate forceps delivery may be associated with facial paralysis.

incidence 0.05 to 0.23%

INFECTIOUS ETIOLOGY

OTITIS MEDIA

Can occur in Acute Suppurative Otitis Media, Chronic Suppurative Otitis Media, Tuberculosis

Acute Suppurative Otitis Media

Facial palsy is due to toxic neuritis, hyperaemia or edema of loose fibrous tissue of the nerve. Most common in children.

Mainstay of treatment is antibiotics and myringotomy. Some patients cortical mastoidectomy is needed.

Chronic Suppurative Otitis Media

Facial palsy can occur in cholesteatoma and non cholesteatoma cases

CHOLESTEATOMA

Palsy is due to erosion of the bony canal by cholesteatoma matrix and then direct pressure on the nerve and effect of various enzymes secreted by matrix

Treatment is Mastoid exploration with facial nerve decompression.

TUBERCULOUS OTITIS MEDIA

In early cases facial palsy due to erosion of the bone by granulation tissue or formation of bony sequestra. In late cases may be due to tuberculoma in the mastoid causing facial palsy

CLINICAL PRESENTATION

- Multiple perforation
- Painless purulent discharge
- Conductive hearing loss
- Pale granulation

The treatment is decompression of the nerve with removal of granulations followed by antituberculous therapy

PATHOPHYSIOLOGICAL MECHANISMS

- Through the fallopian canal dehiscence or physiological canaliculi for neurovascular structures, direct infection of the nerve.
- Fallopian canal erosion due to osteitis and nerve involvement
- Compression of the nerve due to inflammatory edema.
- Secondary thrombosis of the vasa nervorum with ischemia and infarction of the nerve.

INVESTIGATIONS

Imaging studies used for identifying intracranial and intratemporal complications of ASOM and CSOM.

CT scan delineate the fallopian canal erosion

MALIGNANT OTITIS ETERNA

It is defined as potentially life threatening and aggressive infection of the soft tissues of the external canal and surrounding structures that rapidly spreading to periosteum and bone of the skull base.

CLINICOPATHOLOGICAL STAGING

1. Clinical evidence of MOE with infection of soft tissues extending beyond the external canal with negative Technetium99 bone scan
2. Soft tissue infection beyond external canal with positive Technetium99 bone scan
3. As above with cranial nerve paralysis

   3a single

   3b multiple

4. Meningitis, Empyema, Sinus thrombosis or Brain abscess

   infection progress through stage of cellulitis, chondritis, periostitis, osteitis and osteomyelitis. Facial palsy due to spread of infection

through the bony cartilaginous junction and fissure of santorini and to the stylomastoid foramen

Most common organism is pseudomonas

Predisposing factors

Diabetes mellitus, neoplasm of temporal bone and HIV

CLINICAL FEATURES

Pain, exudates, granulations and edema of external canal

DIAGNOSIS

Technetium 99 radionucleide Bone scan detect bony involvement. Because it is absorbed by osteoclasts and osteoblats. It is remain positive upto nine months.

Gallium 67 bone scan detect infection. because it is absorbed by leukocytes

CT to know about localization of disease extent

MRI to differentiate between malignant and inflammatory process

MANAGEMENT

Control of diabetes

Ciprofloxacin 750mg twice daily orally for a period of 6-8 weeks

Surgical debridement if needed

VARICELLA ZOSTER VIRUS INFECTION

(RAMSAY HUNT SYNDROME)

Peripheral facial palsy associated with erythematous vesicular rash on the ear or in the mouth. The cause is reactivation of VZV in geniculate ganglion.

Incidence of facial palsy in adults is 3 to 12%

In children 5%

SYMPTOMS AND SIGNS

- Severe persisting and excruciating ear pain
- Vesicular eruptions in the EAC
- Facial palsy
- Sensorineural hearing loss

DIAGNOSIS

Confirmed by rising titres of vzv antibodies

Complete recovery occur in 10% of patients. In 30-50% of patients persistent weakness is observed. Improvement of palsy occur if acyclovir and prednisolone is started within three days of onset. They reduce post herpetic neuralgia, vertigo and otalgia. Surgical decompression is not indicated.

Other viral illness causing facial palsy

Gullian Barre Syndrome

EBV virus

HIV

INFLAMMATORY DISORDERS OF FACIAL NERVE

- SARCOIDOSIS

  Facial palsy developed either isolated or in association with uveitis and parotiditis (heerfordt's diseases)
- WEGENER'S GRANULOMATOUS
- LYME'S DISEASE

NEOPLASMS

Incidence of facial palsy due to neoplasm is 5%. Most of the facial palsy caused by extrinsic compression by tumors in and around temporal bone. In adults skull base neoplasm and metastasis are common. In children haematological malignancies are common

Most common is squamous cell carcinoma of the head and neck.

Tumors of temporal bone and metastasis from nasopharynx can involve the facial nerve.

Common sign is progressive facial paralysis. Early diagnosis by high index of suspicion. Involvement of other cranial nerves and recurrent facial palsy also common.

In Vestibular schwannoma facial palsy is uncommon and if present it indicates advanced stage. Facial palsy due to extrinsic compression or may be due to local vascular compromise by tumor enlargement within the IAC,Nerve sheath tumors meningioma, schwannoma and hemangioma present with facial palsy in early stage. Management is tumor resection and grafting.

SURGICAL APPROACHES

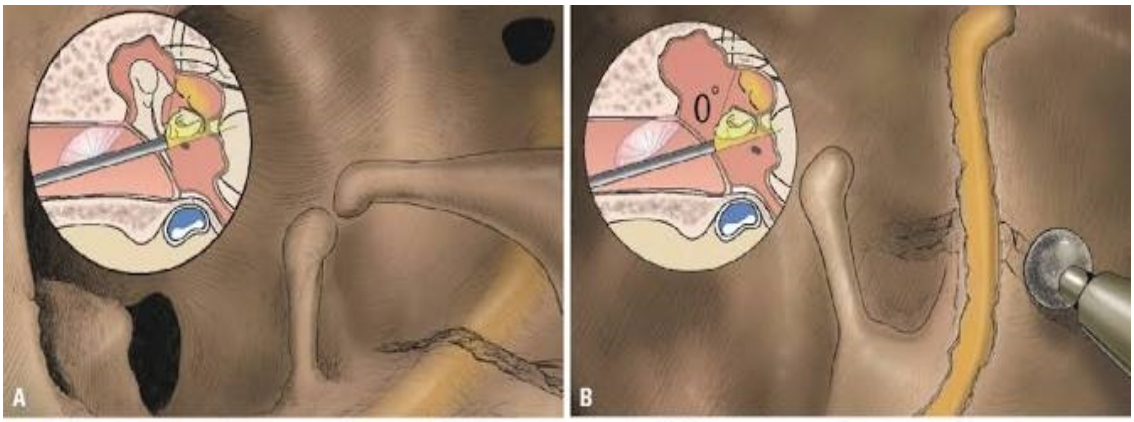
ENDOSCOPIC TRANSCANAL APPROACH

- Incision is made in the external auditory canal from 11 to 7 o' clock position and tympanomeatal flap elevated and expose the scutum
- Tympanic membrane separated from the malleus and expose medial wall of the middle ear
- Posterior canal wall curetted without injuring chorda tympani nerve to access anterior epitympanum
- Incus removed to expose the nerve from geniculate ganglion to second genu
- Cog and cochleariform process identified. These are the land marks for 1st genu

- Lateral semicircular canal identified posterior and superior to the second genu. This is the posterior limit of the dissection
- Then facial nerve was decompressed from geniculate ganglion to second genu. If needed upto stylomastoid foramen. Gelfoam placed to close the nerve
- Ossicular reconstruction was done.
- underlay tympanoplasty was done
- Cartilage reconstruction was done
- Tympanomeatal flap repositioned

Advantages

Cosmetical. No scar



TRANSMASTOID APPROACH

1) CANAL WALL DOWN PROCEDURE

- It provides exposure of tympanic and mastoid segment of facial nerve
- Limited access to labyrinthine segment and geniculate ganglion
- Lateral semicircular canal and digastrics ridge are important landmarks for identification
- Conductive hearing loss may occur

SURGICAL PROCEDURE

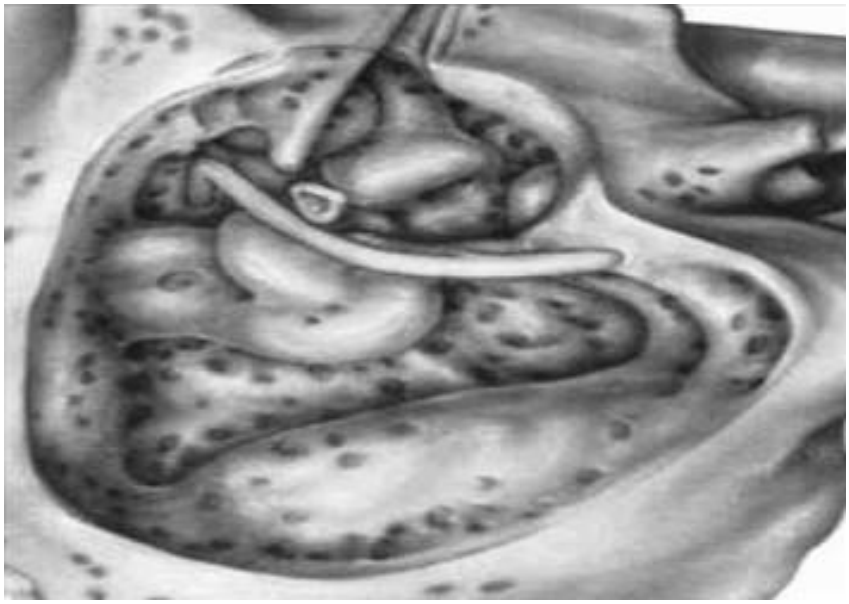
- Modified William wilde post aural skin incision made. subcutaneous tissue elevated
- periosteum is elevated from mastoid after making T shaped incision in periosteum
- Maceven's triangle is identified and drilled
- Suprameatal triangle or MAC EVEN'S triangle boundaries

  Posterosuperior part of EAC anteriorly

  Suprameatal crest superiorly

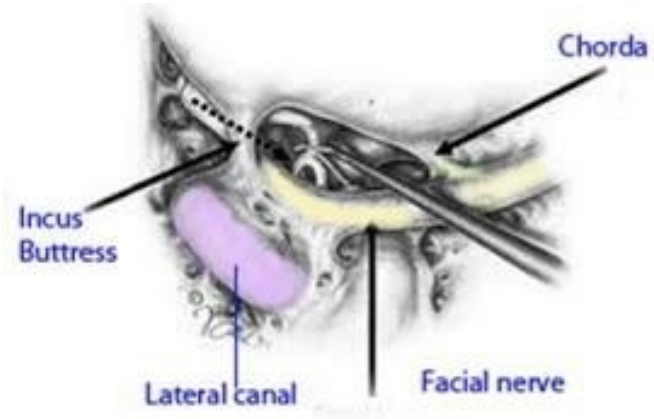
  Tangential line connecting these two points posteriorly
- Complete mastoidectomy done.
- Posterior canal wall is reduced.

- Bridge that is part of posterior canal wall lateral to aditus and antrum is removed.
- Anterior buttress that is bony projection at the junction of tegmen and anterior bony canal is removed.
- Posterior buttress that is bony projection at the junction of floor of EAC with posterior canal wall is removed.
- Facial ridge that is part of posterior meatal wall lateral to the mastoid segment of facial nerve is reduced.
- Then proceed to removal of cholesteatoma by careful dissection in CSOM
- Facial nerve decompression done.



POSTRIOR TYMPANOTOMY - OPERATIVE PROCEDURE

- Post auricular incision is made. Temporalis fascia retracted upward to expose posterior root of zygoma and suprameatal triangle.
- Complete mastoidectomy done.
- Facial recess opened and exposes tympanic portion of nerve.
- Inferiorly facial recess can be expanded by sacrificing chordatympani nerve. This approach is known as extended facial recess appraoach.
- Using diamond burr nerve is delineated and leaving thin layer of bone over the nerve.
- After exposure egg shell bone over the nerve removed and nerve sheath opened.



MIDDLE CRANIAL FOSSA APPROACH

This method exposes internal auditory canal and labyrinthine segments of facial nerve with preservation of hearing

In combination with transmastoid approach it possible to visualise entire course of the nerve.

PROCEDURE

- Posteriorly based trap door incision made in the hairline above the ear.
- Temporalis muscle is exposed by elevating skin flap.
- Temporal root of zygoma which marks the level of floor of middle cranial fossa is exposed by elevating temporalis muscle
- 4x5 cm bone flap made above temporal root of zygoma and elevated without injuring middle meningeal artery.
- Dura is retracted posterior to anteriorly for preventing injury to an geniculate ganglion and GSPN.
- Drilling is done posterior to arcuate eminence and continued till otic capsule is identified. Then blue line of SCC is seen after removing otic capsule.
- Drilling is done along the petrous ridge. Nearly 180 degree of canal can be exposed.
- Facial nerve can be identified in the anterosuperior part of IAC.
- Then facial nerve followed to geniculate ganglion.

RETROLABYRINTHINE APPROACH

Exposes the facial nerve from brainstem to internal auditory meatus

- Curvilinear incision made 3-4 cm posterior to post auricular crease extending upto mastoid tip.
- Complete mastoidectomy done. In addition bone over the sigmoid sinus is removed from sinodural angle to most inferior aspect of the sinus.
- Jugular bulb identified.
- Posterior semicircular canal and endolymphatic sac are exposed.
- Anteriorly oriented cerebellar dural flap made. But dura over the bony margins, sigmoid sinus and superior petrosal sinuses are left for placement of sutures during closure.
- Sigmoid sinus retracted posteriorly using self retaining retractor.
- Facial nerve and cochleovestibular nerve seen 2-3 mm inferior and parallel to the donaldson's line.
- Dura is closed with 4-0 nylon suture.
- Mastoid cavity obliterated using fat and subcutaneous tissue.
- Post aural wound sutured.

RETROSIGMOID APPROACH

Exposes facial nerve from brainstem and internal auditory meatus.

- Post aural skin incision made 2-3 cm posterior to post auricular crease
- Complete mastoidectomy usually not needed. Instead bone over the sigmoid sinus is removed to identify posterior margin of the sinus.
- Posterior cranial fossa is exposed by doing craniotomy 4cm posterior to the sigmoid sinus.
- Cerebellum is exposed by a curvilinear incision in posterior fossa dura.
- After gentle retraction of cerebellum we can visualise facial and cochleovestibular nerve. For additional exposure posterior lip internal auditory meatus can be removed

NERVE REPAIR

When continuity of nerve is disrupted by traumatic or iatrogenic injury we should attempt to restore its continuity by end to end anastomosis or interposition nerve grafting with greater auricular nerve and sural nerve.

REVIEW OF LITERATURE

Sir Charles bell was described facial nerve anatomy and facial nerve palsy.

Avicenna described causes, management and prognosis of facial nerve palsy

Before sir Charles bell, Stalpart van der wiel first described about Bell's palsy.

In 18th century James douglas described about facial palsy

In 1943 Turner said facial nerve is mostly affected in closed head injuries

In 1976 potter and Braakman said mechanism of delayed facial palsy is not obvious like that of immediate palsy

In 1879 Drobnick did first nerve anastomosis by anastomosing facial nerve to spinal accessory nerve

In 1927, Martin did end to end suturing in fallopian canal

In 1936, Bunnel did first facial nerve graft

Puvanendran, Grove and Turner studied 80 to 90% of delayed facial palsy will spontaneously recovers[53].

Santos et al studied facial palsy is common etiology is Bell's palsy followed by trauma [54].

Chang and Cass studied surgical decompression gives better result if done within 14 days of injury [36].

Ikeda,et al[39] reported duration between onset of facial paralysis and surgery is an important factor determining outcome of facial palsy.

Jin Kim ,Gu- Hyun - Jung[38] said facial palsy can be seen in patients with cholesteatoma and without cholesteatoma . Nowadays facial palsy in SOM is declined due to widespread use of antibiotics. complete removal of cholesteatoma in patients with longer duration of facial paralysis does not give good recovery. They studied surgery is mainstay of treatment extent of disease, with or without cholesteatoma, type of surgery and delay in surgery are prognostic factors affecting outcome of surgery

Joseph and Sperling[55] reported that direct involvement of nerve by bacterial or viral toxins is the common mechanism in ASOM

Zinis[40] reported facial nerve involvement in infectious causes by infections passing through the bony dehiscence or neurovascular communications between the nerve and middle ear.

Makeham TP et al[56] said facial palsy in patients without cholesteatoma gives better outcome than cholesteatoma with facial palsy.

He also reported in ASOM facial palsy gives better results without decompression

Shyam S.Kumar[59] and Alok thakar studied patients with complete palsy of longer duration need static or dynamic reanimation in addition to facial nerve decompression

In 1953 Wallmer described clinical features of tuberculous otitis media

Varty et al[48]. Studied combination of medical therapy and surgical management gives better recovery in tuberculous facial palsy

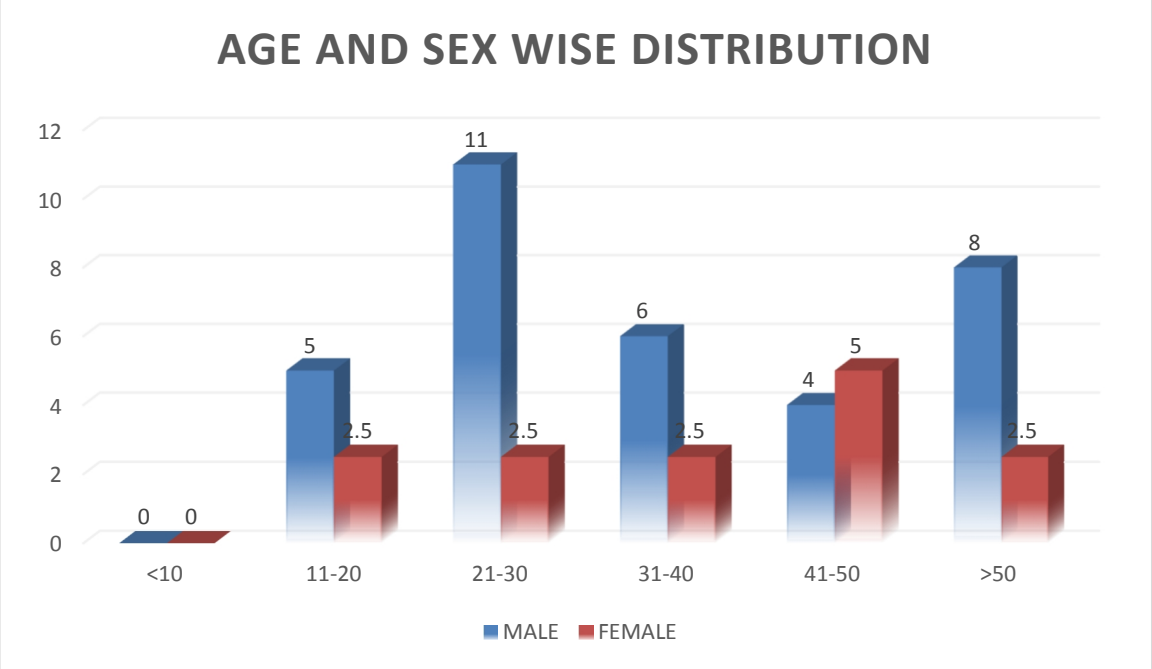
Nicola Quaranta[57] and Marilina Santantonia reported tuberculous otitis media with facial palsy can be treated by antituberculous treatment. Surgery reserved only for complications.

Zainine R, et al[58] studied patients with ramsay hunt syndrome has poor prognosis than that of bell's palsy.

RESULTS

1)AGE AND SEX WISE DISTRIBUTION

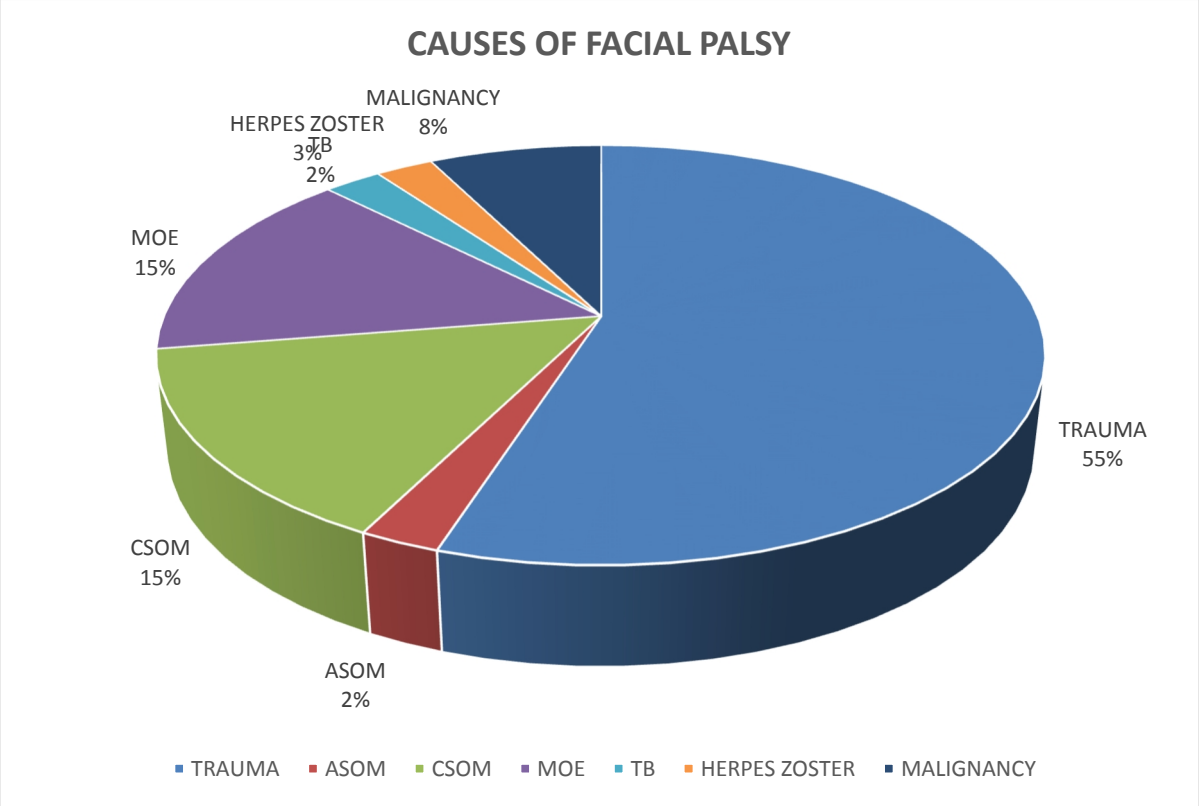
AGE	MALE	%	FEMALE	%
<10 YEARS	0	0	0	0
11 -20	5	12.5	1	2.5
21-30	11	27.5	1	2.5
31-40	6	15	1	2.5
41-50	4	10	2	5
>50	8	20	1	2.5
TOTAL	34	85	6	15



A total of 40 cases are included in this study during the study period .Among the 40 cases 34 cases (85%) are male and 6 cases(15%) are female. commonest age group is 21-30years.

2) CAUSES OF FACIAL PALSY

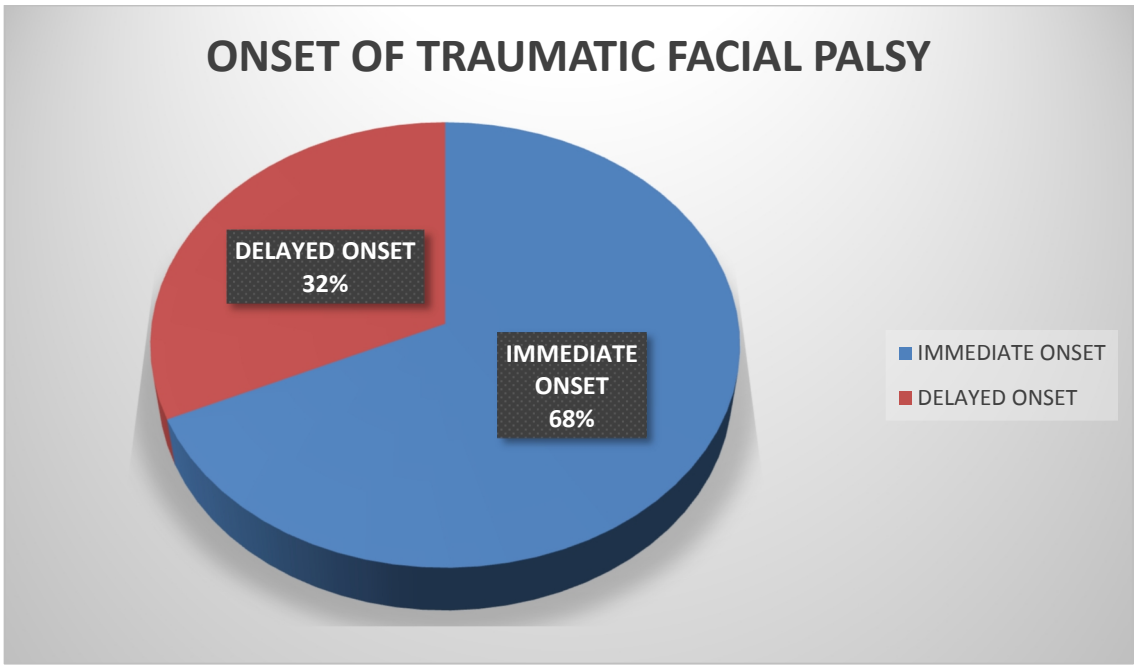
CAUSES	No. of patients	%
TRAUMA	22	55
ASOM	1	2.5
CSOM	6	15
MOE	6	15
TB	1	2.5
HERPES ZOSTER	1	2.5
MALIGNANCY	3	7.5
TOTAL	40	100



Commonest cause of facial palsy is trauma in this study. Among the 40 cases 22 cases(55%) are due to trauma following road traffic accident. Next common cause is suppurative otitis media. 7 (17.5%)cases are due to suppurative otitis media including both ASOM and CSOM. Malignant otitis externa contributes 6 cases(15%) of facial palsy. Least common are both tuberculous otitis media (2.5%) and herpes zoster (2.5%).

3)ONSET OF TRAUMATIC FACIAL PALSY

ONSET OF PRESENTATION	NO. OF PATIENTS	%
Immediate	15	68.18
Delayed	7	31.82
TOTAL	22	100



Among the 22 cases of traumatic facial palsy 15 cases (68.18%) had immediate onset of facial palsy following trauma . Other 7cases (31.82%) had delayed onset of facial palsy.

4)LEVEL OF INJURY IN TRAUMATIC FACIAL PALSY

LABYRINTHINE SEGMENT	1ST GENU	TYMPANIC PART	2ND GENU	MASTOID SEGMENT	EXTRATEMPORAL
0	2	4	8	6	2

5)TYPE OF FRACTURE IN INTRATEMPORAL INJURY

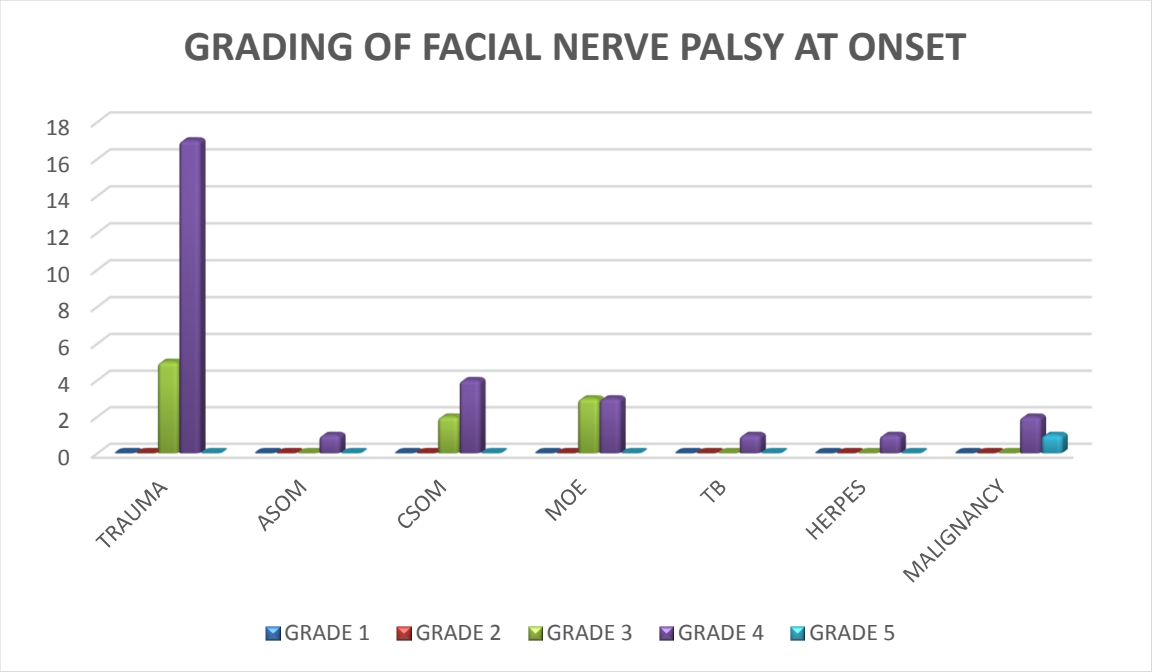
LONGITUDINAL	20
TRANSVERSE	0
MIXED	0
TOTAL	20

Among the 22 patients, 20 cases had injury at the intratemporal course of the nerve. Most common site of injury is at the level of second genu. Only 2 cases had injury at the extratemporal course of the nerve.

Among the 20 cases with intratemporal injuries all had longitudinal type of fracture.

6)GRADING OF FACIAL NERVE PALSY AT ONSET

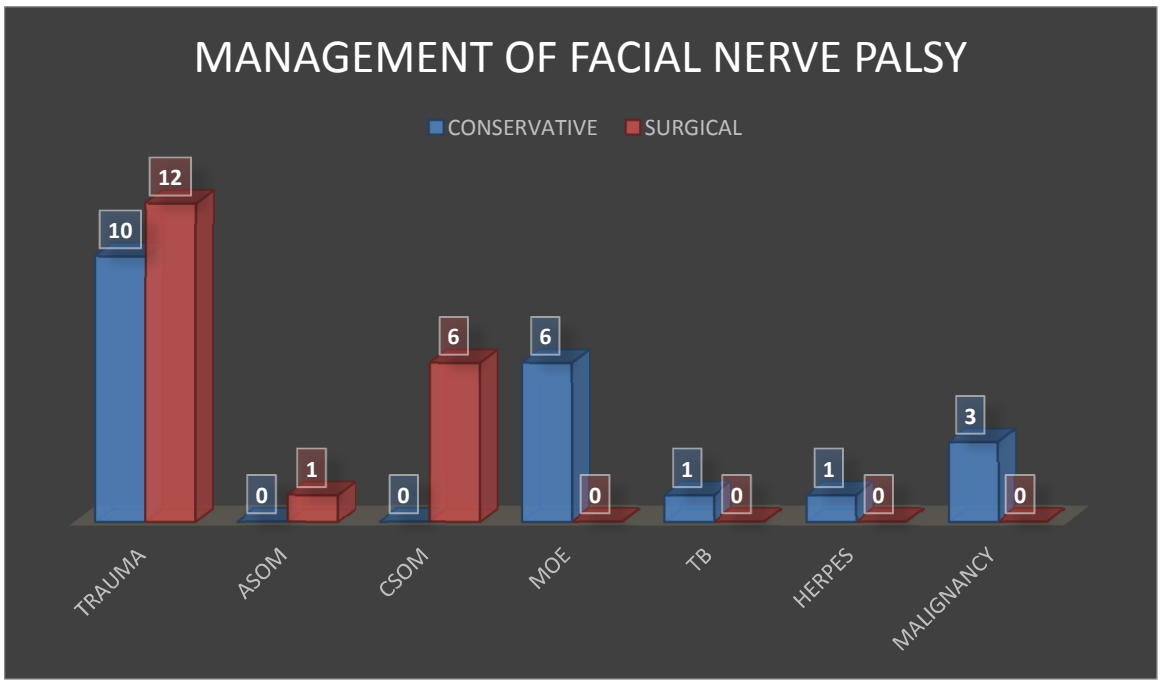
GRADE	NO. OF PATIENTS
GRADE **I**	0
GRADE **I I**	0
GRADE **III**	10
GRADE **IV**	29
GRADE **V**	01
GRADE **VI**	0



Most of the patients presented with grade 4 facial palsy. Among the 22 cases of traumatic facial palsy 17 cases were presented with grade 4 facial palsy. Only 5 cases were presented with grade 3 facial palsy. Among the 6 patients with chronic suppurative otitis media 4 patients presented with grade 4 palsy and 2 cases presented with grade 3 palsy .In Malignant otitis externa 3cases presented with grade 4 facial palsy. 75% of patients presented with grade 4 and above and 25% presented with grade 3 palsy.

7)MANAGEMENT OF FACIAL NERVE PALSY

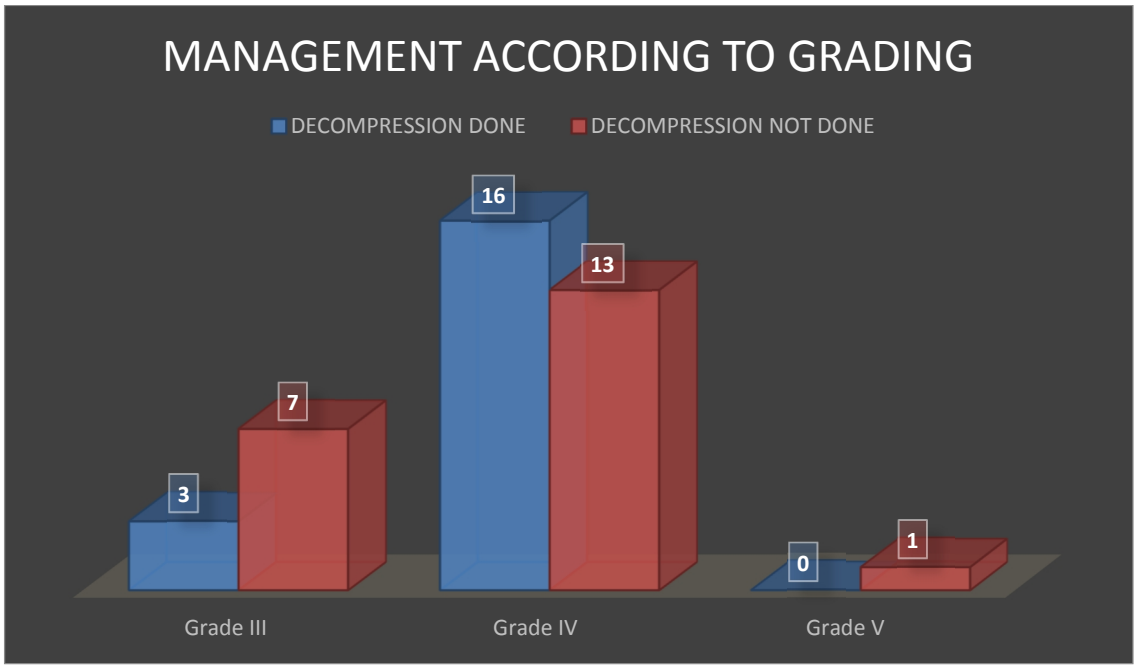
CAUSES	CONSERVATIVE	%	SURGICAL	%
TRAUMA	10	25	12	30
ASOM	-	-	1	2.5
CSOM	-	-	6	15
MOE	6	15	-	-
TB	1	2.5	-	-
HERPES	1	2.5	-	-
MALIGNANCY	3	7.5	-	-
TOTAL	21	52.5	19	47.5



Among the 40 patients 19 patients (52.5%)underwent facial nerve decompression and 21patients are treated with appropriate treatment without facial nerve decompression. In case of trauma out of 22(55%) patients 12 patients (30%) underwent facial nerve decompression. In acute and chronic suppurative otitis media all patients were taken up for surgery.

8)MANAGEMENT ACCORDING TO GRADING

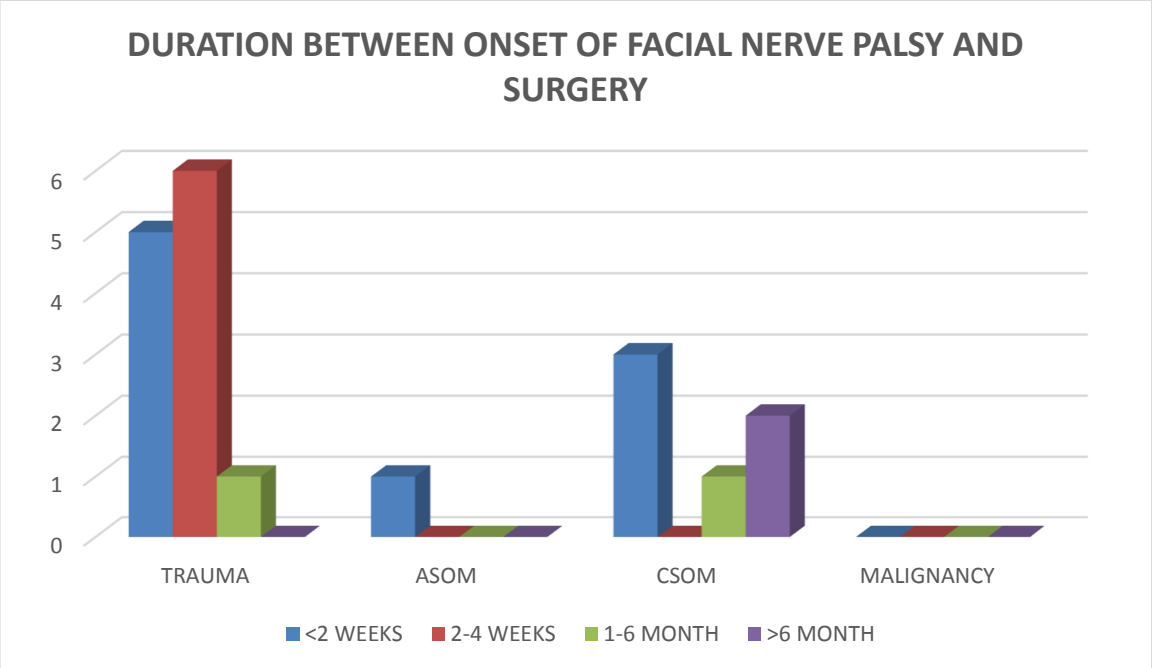
GRADE	DECOMPRESSION DONE	DECOMPRESSION NOT DONE
III	3	7
IV	16	13
v	-	1



Among the 19 cases(47.5%) who underwent surgey,16 cases(40%) of cases are presented with grade 4 and above. 3 cases (7.5%) are presented with grade 3 palsy. Among the 21 cases(52.5%) underwent conservative management 7cases (17.5%) are presented with grade 3 palsy.14 cases (35%) are presented with grade 4 palsy.

9)DURATION BETWEEN ONSET OF FACIAL NERVE PALSY AND SURGERY

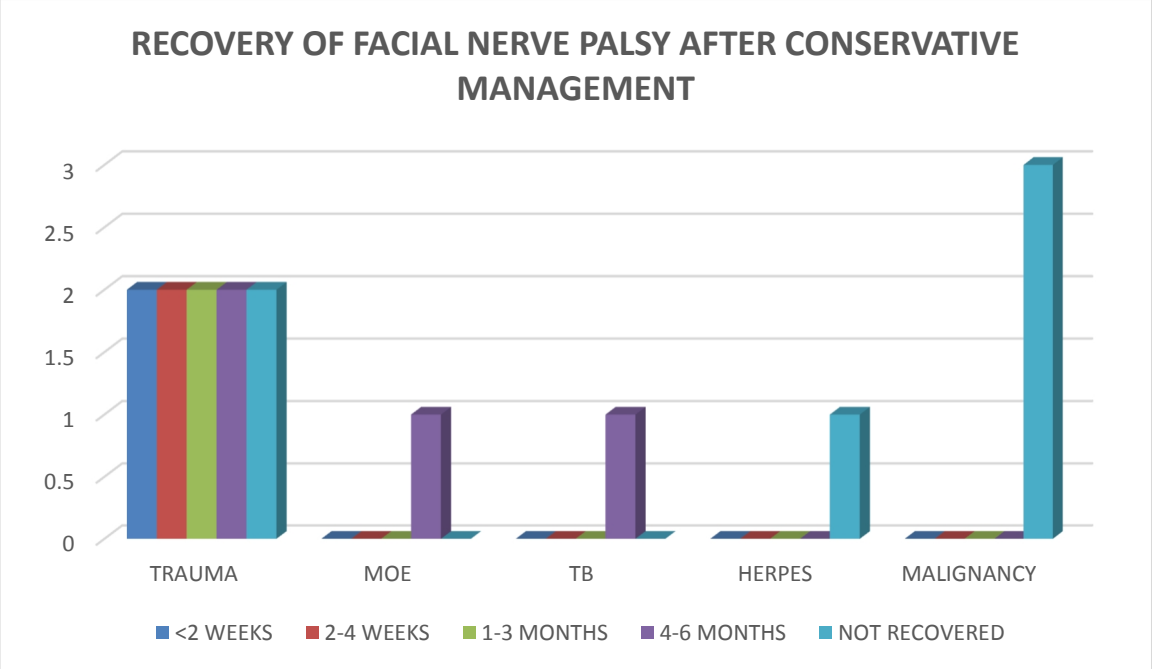
CAUSES	<2 WEEKS	2-4 WEEKS	1-6 MONTHS	>6 MONTH
TRAUMA	5	6	1	0
ASOM	1	0	0	0
CSOM	3	0	1	2
MALIGNANCY	0	0	0	0
TOTAL	9	6	2	2



Among the 19 patients (47.5%) ,9 patients(22.5%) taken for surgery within 2 weeks and 6 patients taken for surgery within 2 to 4 weeks. Only 2 patients taken for surgery more than 6 months after onset of palsy

9) OUTCOME OF FACIAL NERVE PALSY AFTER CONSERVATIVE MANAGEMENT

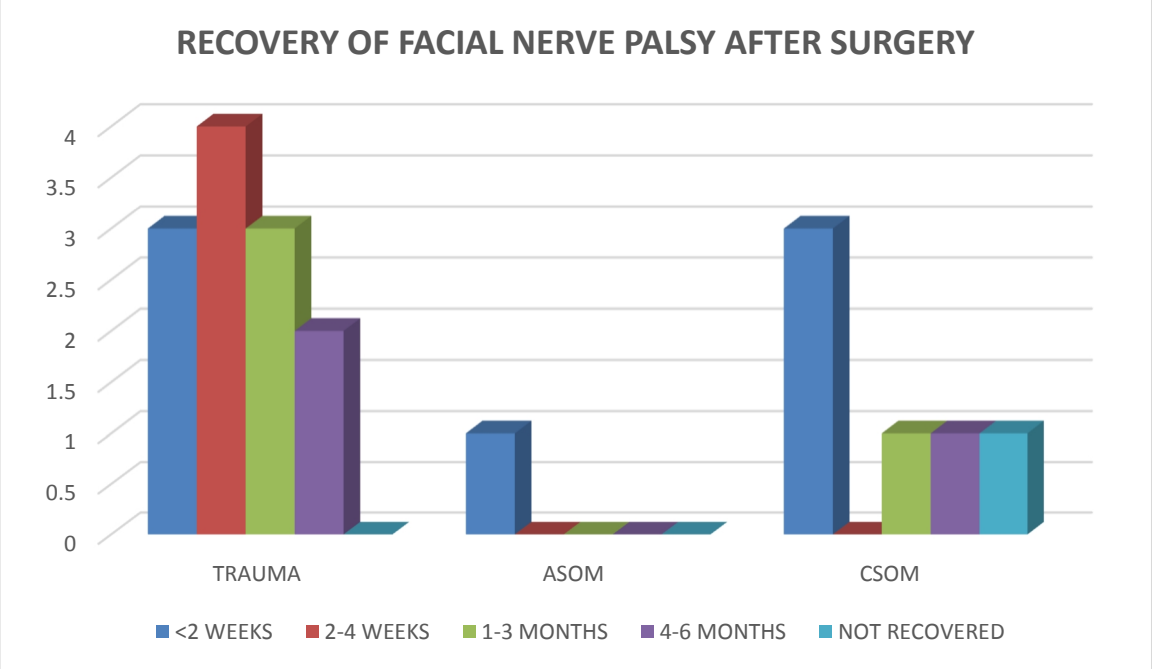
DURATION	GRADE	TRAUMA	MOE	TB	HERPES	MALIGNANCY	TOTAL
< 2 weeks	**I**	2	-	-	-	-	2
2-4 weeks	**I**	2	-	-	-	-	2
1-3 months	**I**	2	2	-	-	-	4
4-6 months	**I**	2	1	1	-	-	4
	II	-	2	-	-	-	2
Not recovered	-	2	1	-	1	3	7



Among the 21 cases (52.5%) ,14 cases(35%) are recovered after conservative management. 7 cases (17.5%) did not recover. In traumatic FP out of 10 patients 5 patients with grade 3 and 3 patients with grade 4 completely recovered after conservative management. Patient with tuberculous otitis media recovered after ATT. In MOE 3 patients with incomplete palsy are recovered . In patients of MOE with grade 4 palsy 2 patients are recovered and one patient not recovered.

10)RECOVERY OF FACIAL NERVE PALSY AFTER SURGERY

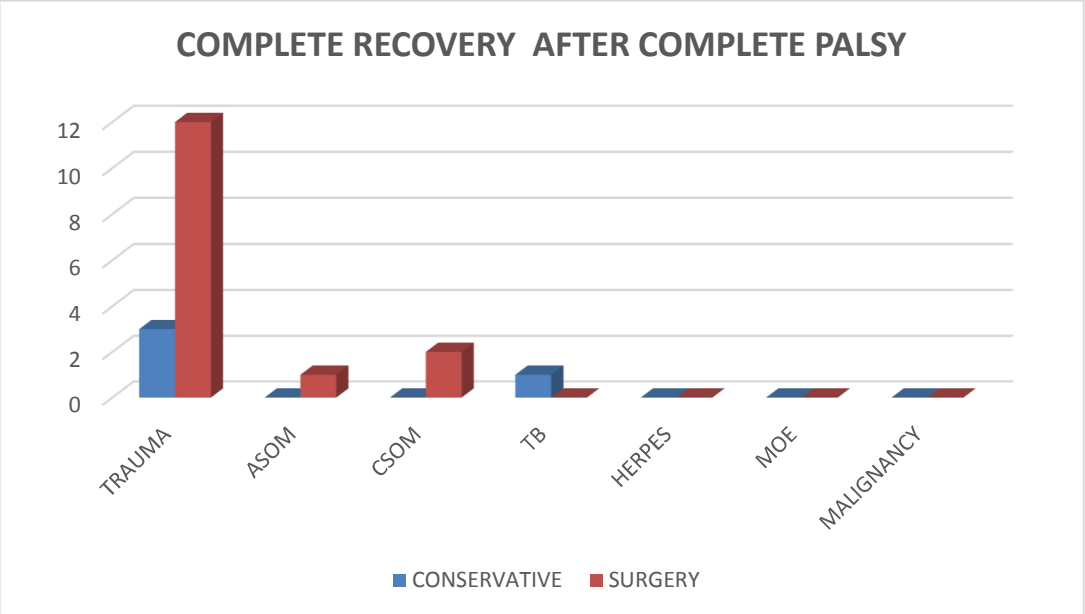
TIME OF RECOVERY AFTER SURGERY	GRADE	TRAUMA	ASOM	CSOM	TOTAL
<2 WEEKS	**I**	3	1	3	7
2-4 WEEKS	**I**	4	-	-	4
1-3 MONTHS	**I**	3	-	1	4
4-6 MONTHS	**I**	2	-	1	3
NOT RECOVERED	-	-	-	1	1



After surgical management out of 19 patients(47.5%) 18 patients(45%) are recovered. In traumatic facial palsy out of 12 cases taken for surgery all cases are completely recovered .In chronic suppurative otitis media out of 6 cases, 5 patients recovered. One case did not recover. In acute suppurative otitis media with facial palsy the patient completely recovered after surgery.

11) COMPLETE RECOVERY AFTER COMPLETE PALSY

CAUSE	CONSERVATIVE	SURGERY
TRAUMA	3	12
ASOM	0	1
CSOM	0	2
TB	1	0
HERPES	0	0
MOE	0	0
MALIGNANCY	0	0
TOTAL	4	15



Among the 16 patients with grade 4 palsy who underwent surgery 15 cases recovered to grade 1 after surgery.

DISCUSSION

Lee shahinian[32] studied the chance of return of facial nerve function in traumatic facial paralysis is enhanced by early surgical decompression of the nerve. In my study out of 55% of traumatic facial palsy 42.5% of patients presented with complete palsy. Out of 42.5% of patients 30% of patients underwent surgery. All patients recovered to grade1 following surgical decompression of the nerve.

Marchoni[65] studied tympanic portion of the facial nerve can be visualised from geniculate ganglion to second genu by endoscopic approach.

Kahinga AA[52] studied 2 patients of traumatic facial palsy who underwent endoscopic transcanal decompression. Both recovered within 6 months and also there is improvement of preoperative hearing impairment in his study. He concluded that this approach is useful in cases of traumatic facial palsy involving tympanic part from Geniculate ganglion to second genu and this surgery does not affect hearing.

In this study out of 12 patients, 11 underwent transcanal endoscopic facial nerve decompression. According to this study the nerve can be exposed from Geniculate ganglion upto stylomastoid foramen by transcanal approach. Only one patient underwent modified radical mastoidectomy.

In this study out of 11 patients who underwent transcanal endoscopic facial nerve decompression, in nine patients preoperative hearing is normal. No conductive deafness. Post operatively in these nine patients hearing is same as pre operative hearing level. But in other 2 patients there is preoperative conductive deafness. Out of these 2 patients in one patient hearing improved. so according to my study transcanal facial nerve decompression does not affect hearing.

Dragolijub popovic[34] also studied facial nerve decompression is more effective in cases of traumatic facial palsy. Sillman JS and Nipako JK studied those who underwent surgery showed grade 1 recovery. Those who did not undergo surgery showed incomplete (grade 2 or 3) recovery. In my study also facial nerve decompression in traumatic facial palsy gives complete recovery in all cases who have underwent surgery.

Chang CY[36] studied decompression surgery gives better outcome if done within 14 days of injury. In my study patients with traumatic facial palsy who were taken for decompression even after 14 days also had grade 1 recovery . In this study out of 30% of patients who underwent surgery, only 12.5% of patients were taken for surgery within 14 days of injury. Other 17.5% of patients were taken for surgery after 14 days of injury. So according to this study facial nerve decompression can be taken irrespective of duration of facial palsy.

Devang P.gupta [35] studied commonest cause of facial palsy after excluding Bell's palsy is trauma. Articles in Archives of Hellenic medicine studied commonest cause of facial palsy is trauma followed by otitis media. In this study also common cause is trauma.55% of patients developed facial paralysis following trauma.

Med J Armed forces india[37], 2017 published patients with facial palsy due to otitis media showed 100% recovery after clearance of disease and decompression. In this study out of 6 cases 5 cases showed grade 1 recovery.

Jin Kim, Gu-Hyun Jung[38] study, patients with cholesteatoma and longer duration of facial palsy, prognosis is poor and in patient shorter duration of facial palsy without cholesteatoma prognosis is better after surgery. In this study 4 patients without cholesteatoma who had facial palsy recovered after surgery in spite of one patient having longer duration of facial palsy. Out of two patients with cholesteatoma and facial palsy one patient with longer duration of complete facial palsy did not recover after surgery and one patient with cholesteatoma and shorter duration of facial palsy recovered after surgery.

Ikeda M and Nakazota[39] also studied patients with cholesteatoma prognosis of facial palsy is poor

Zinis LO, gamba P,Balzanelli[40] studied in patients with acute suppurative otitis media and facial palsy can be treated with antibiotics and myringotomy and patients with coalescent mastoiditis need mastoidectomy. Facial nerve decompression is not necessary. In my study patient with acute suppurative otitis media and coalscent mastoiditis is recovered after mastoidectomy without facial nerve decompression.

R.B.Sardesai and T.Krishnakumar[41] studied early diagnosis of malignant otitis externa and treatment with local excision and debridement in addition to antibiotic treatment give better results.

Karthick Shameena, Veena B.ganga[51] studied 8 patients with malignant otitis externa with facial nerve palsy. All were treated with antipseudomonal antibiotics and maintained strict glycemic control. Medical management gives a better results and surgery has lesser role in their study. But 2 patients had residual palsy. In my study 6 patients are studied. Out of 6, 3 patients with incomplete palsy had complete recovery after medical management and strict glycemic control. And in 3 patients with complete palsy debridement has done. Out of these 3, 2 had grade 2 recovery.

According to Nyrop[43] patients with early malignancy survival rate was good. In cases with advanced malignancy survival was poor.

According to Higgins et al[47], involvement of facial palsy associated with poor outcome. In this study none of the patients had recovery

Ryu EW and LEE Hy[45] studied prognosis of Ramsay hunt syndrome was poor. In my study one case of Ramsay hunt syndrome is reported and patient had no recovery. But Rafeal da costa Monsanto[46] studied higher rate of recovery if diagnosed early and treated with antiviral and steroids

Varty et al[48] both surgery and medical therapy gives better results in tuberculous otitis media. In my study medical therapy gives grade 1 recovery. Carl M Krisch, John wehner[49] studied medical therapy with antituberculous treatment is effective. Surgery rarely needed.

CONCLUSION

Patients with facial palsy were evaluated and managed according to their etiology and grading of palsy at onset.

- Most commonest age group of facial nerve paralysis is 20-40 years Most common in males.
- Most common cause is trauma followed by otitis media in case of traumatic facial paralysis facial nerve decompression gives complete recovery (grade1) even in patients with grade 4 palsy.
- Patients with incomplete palsy can be managed conservatively
- Patients with chronic suppurative otitis media with facial palsy needed surgery to clear the disease regardless of grading.
- Chronic suppurative otitis media without cholesteatoma with facial palsy has good prognosis than chronic suppurative otitis media with cholesteatoma
- In chronic suppurative otitis media with cholesteatoma with shorter duration of facial palsy has good prognosis.
- Patients with incomplete palsy will recover earlier than patients with complete palsy.
- In tuberculous otitis media with facial palsy recovery of facial palsy is possible after completion of course of antituberculous treatment.
- In herpes zoster oticus prognosis of facial nerve palsy was poor.

- In malignant otitis externa patients with incomplete palsy can improve after glycemic control and medical management. Patients with complete palsy may need debridement in addition to the above.
- In malignancy prognosis of facial palsy is poor.

PATIENT WITH TRAUMATIC FACIAL NERVE PALSY

PRE OPERATIVE CLINICAL PICTURE

POST OPERATIVE CLINICAL PICTURE

PATIENT WITH TRAUMATIC FACIAL NERVE PALSY

PRE OPERATIVE CLINICAL PICTURE

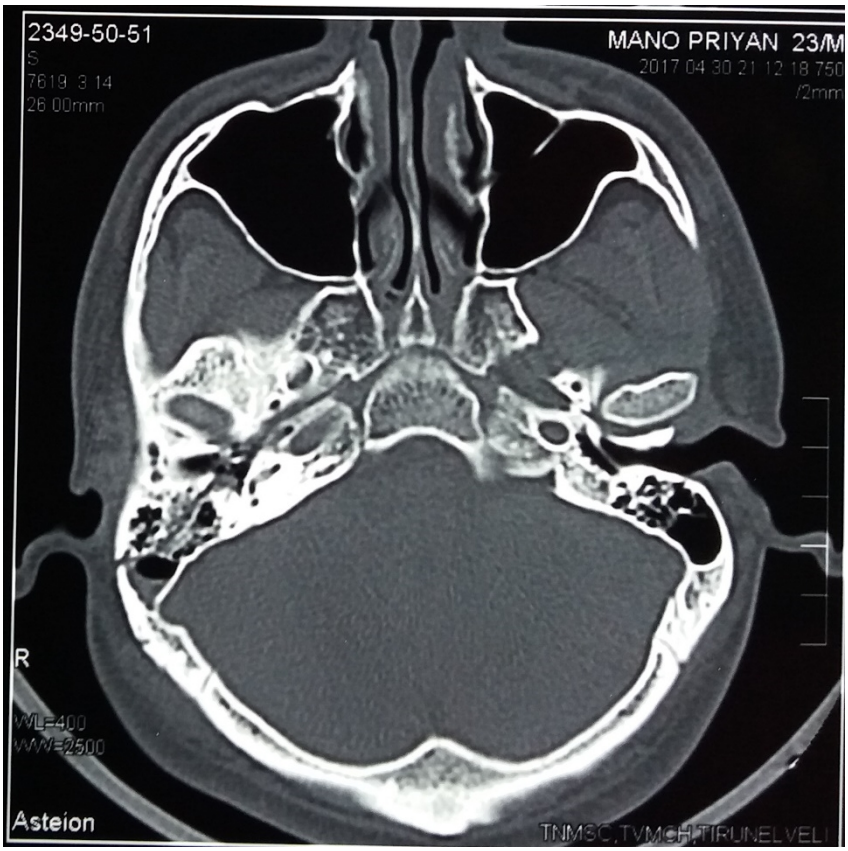
POST OPERATIVE CLINICAL PICTURE

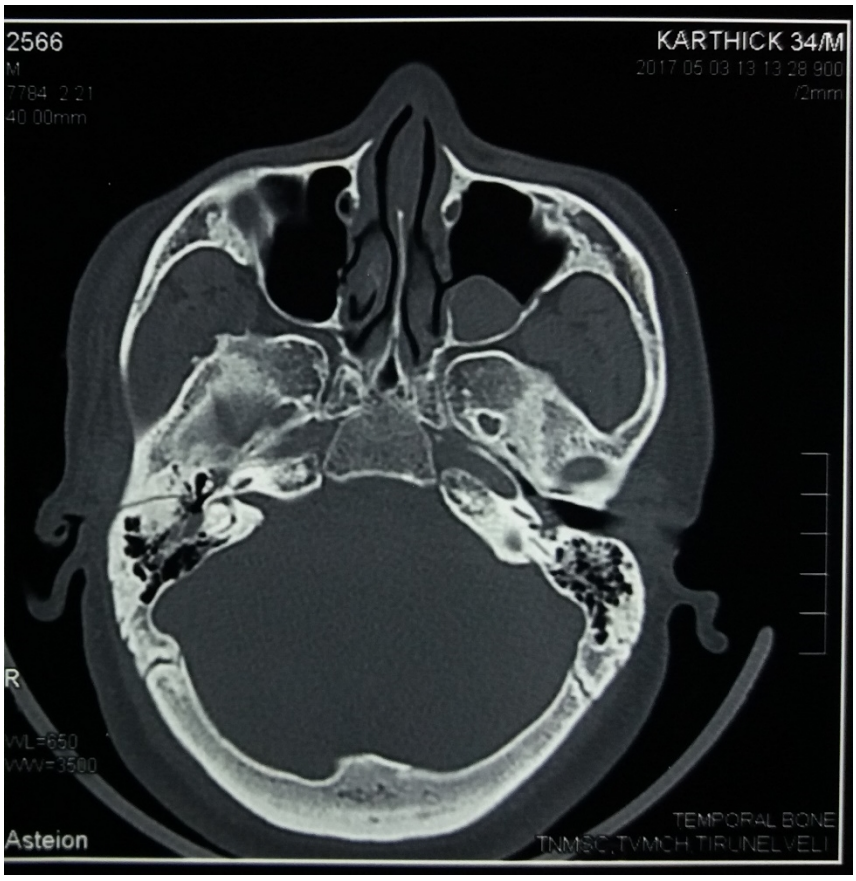
PATIENT WITH TRAUMATIC FACIAL NERVE PALSY

PRE OPERATIVE CLINICAL PICTURE

POST OPERATIVE CLINICAL PICTURE

CT SCAN SHOWING LONGITUDINAL FRACTURE LINE





PATIENT WITH CSOM AND FACIAL NERVE PALSY

PRE OPERATIVE CLINICAL PICTURE

POST OPERATIVE CLINICAL PICTURE

PATIENT WITH TRAUMATIC FACIAL NERVE PALSY

PRE OPERATIVE CLINICAL PICTURE

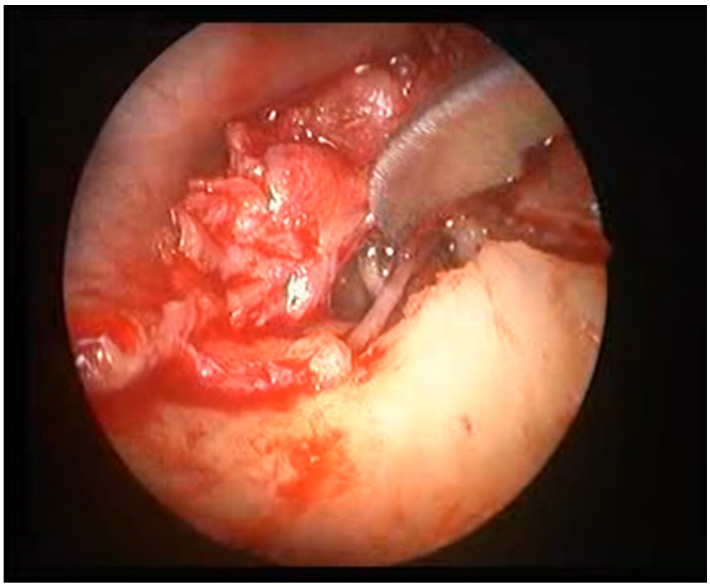
POST OPERATIVE CLINICAL PICTURE

PATIENT WITH TRAUMATIC FACIAL NERVE PALSY

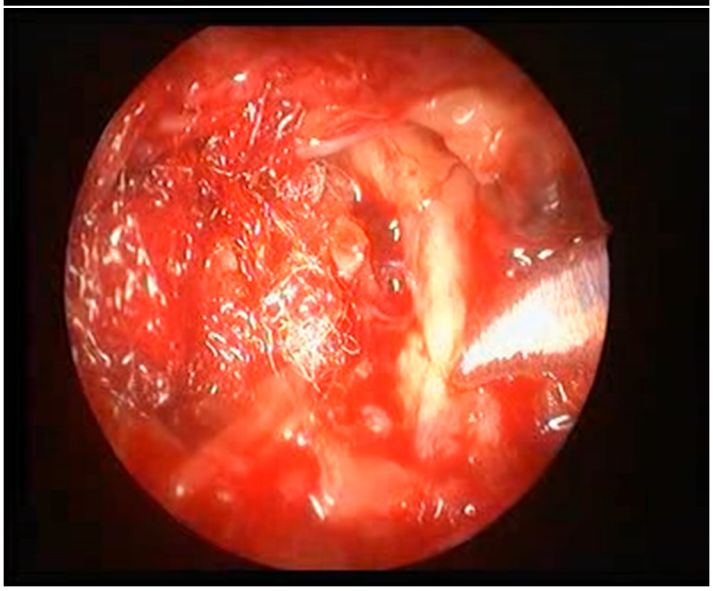
PRE OPERATIVE CLINICAL PICTURE

POST OPERATIVE CLINICAL PICTURE

PER OPERATIVE PICTURE SHOWING FRACTURE LINE



PICTURES SHOWING FACIAL NERVE



PATIENT WITH CSOM AND FACIAL NERVE PALSY

PRE OPERATIVE CLINICAL PICTURE

PICTURE SHOWING NERVE GRAFTING

POST OPERATIVE CLINICAL PICTURE

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PROFORMA

Name
Gender
Age
I.P.No
D.O. Injury if Present
D.O.A
D.O.S
D.O.D
Socio Economic Status
Address

Symptoms

1. Discharge	Present / Absent
2. Type of Discharge	Mucoid / Mucopurulent / Watery
3. Bleeding	Present / Absent
4. Ear pain	Present / Absent
5. Ear blockage	Present / Absent
6. Hard of Hearing	Present / Absent
7. Tinnitus	Present / Absent
8. Onset of injury if present	Delayed / Immediate
9. Facial asymmetry	Present / Absent
10. Dryness of eyes	Present / Absent
11. Incomplete closure of eye	Present / Absent
12. Angle of mouth deviation	Present / Absent
13. Dribbling of saliva	Present / Absent
14. Noise intolerance	Present / Absent
15. Loss of taste	Present / Absent

GENERAL EXAMINATION

Conscious
Anaemic or not
Pulse rate, BP

LOCAL EXAMINATION

EAR	RIGHT	LEFT
pinna		
Preauricular area		
Postauricular area		
External Auditory Canal		
Tympanic Membrane		
Tragal tenderness		
Mastoid tenderness		
Vestibular function		
Fistula sign		

FACIAL NERVE

Facial Symmetry / Asymmetry	
Forehead Wrinkles	Present / Absent
Complete closure of eyes	Present / Absent
Nasolabial fold	Present / Absent
Deviation of Angle of Mouth	Present / Absent

TUNIC FORK TEST

Rinne's

Weber

Absolute Bone Conduction Test

TOPODIAGNOSTIC TESTS

Schirmer Test

Stapedial reflex

Taste

PROVISIONAL DIAGNOSIS

INVESTIGATIONS

Pure Tone Audiometry

CT Temporal Bone

Nerve conduction study if needed

TREATMENT

Conservative

Surgical

S.No	Name	Age/Sex	IP no	Cause	Duration	Grade of onset	Management	Period of recovery	Recovery grade	
1	Jebastin	18 M	67190	Trauma	delayed	GRADE III	CONSERVATIVE	3months	GRADE I	
2	Petchiappan	16 M	69306	CSOM	10days	GRADE III	SURGERY	immediate	GRADE I	
3	Muthusamy	82 M	10924	MOE	1month	GRADE IV	CONSERVATIVE	3months	GRADE II	
4	Ganapathy	42 M	53289	TB	2months	GRADE IV	CONSERVATIVE	6months	GRADE I	
5	Rathinam	80 M	42167	MOE	1month	GRADE III	CONSERVATIVE	6months	GRADE I	
6	Ramar	40 M	58313	HERPES ZOSTER	1week	GRADE IV	CONSERVATIVE		NOT RECOVERED	
7	Geetha	42 M	59327	malignancy	2months	GRADE IV	CONSERVATIVE		NOT RECOVERED	
8	Kaleeshwaran	20 M	21643	CSOM	10months	GRADE IV	SURGERY	3months	GRADE I	
9	Sahaya mary	32 M	43007	CSOM	15days	GRADE IV	SURGERY	2weeks	GRADE I	
10	Arumugam	58 M	58193	MOE	1month	GRADE III	CONSERVATIVE	4months	GRADE I	
11	Subramaniyan	35 M	2301	Trauma	immediate	GRADE IV	SURGERY	3months	GRADE I	
12	Varadharajan	26 M	23072	Trauma	immediate	GRADE IV	SURGERY	6months	GRADE I	
13	Ramesh	30 M	67978	Trauma	delayed	GRADE IV	SURGERY	1month	GRADE I	
14	Selvam	30 M	54678	Trauma	delayed	GRADE IV	SURGERY	1month	GRADE I	
15	Manithai	55 F	63489	malignancy	2months	GRADE V	CONSERVATIVE		NOT RECOVERED	
16	Roja banu	18 F	25645	Trauma	immediate	GRADE IV	SURGERY	immediate	GRADE I	
17	Esakkimuthu	35 M	54445	Trauma	immediate aft inj	GRADE IV	SURGERY	6months	GRADE I	
18	Mohammed ibrahim	71 M	62674	MOE	2weeks	GRADE III	CONSERVATIVE	6months	GRADE I	
19	Manikandan	3 Mch	38746	CSOM	2months	GRADE III	SURGERY	immediate	GRADE I	
20	Chermadurai	45 M	48372	Trauma	immediate	GRADE III	CONSERVATIVE	6months	GRADE I	
21	Arunachalam	45 M	54829	Trauma	immediate	GRADE IV	CONSERVATIVE		NOT RECOVERED	
22	Murugan	57 M	61439	Trauma	immediate	GRADE IV	SURGERY	2weeks	GRADE I	
23	Rajadeepan	32 M	62674	Trauma	delayed	GRADE IV	SURGERY	immediate	GRADE I	
24	Nainar	68 M	63218	MOE	1month	GRADE IV	SURGERY	4months	GRADE II	
25	Pandaram	65 M	53217	MOE	3months	GRADE IV	SURGERY		NOT RECOVERED	
26	Paulraj	30 M	30389	Trauma	delayed	GRADE III	CONSERVATIVE	6months	GRADE I	
27	Esakkiraja	28 M	23581	Trauma	delayed	GRADE IV	SURGERY	1month	GRADE I	
28	Arumuga nainar	28 M	17325	Trauma	immediate	GRADE IV	CONSERVATIVE	2weeks	GRADE I	
29	Karthick	28 M	68213	Trauma	immediate	GRADE IV	CONSERVATIVE	3months	GRADE I	
30	Suseela	45 F	73965	Trauma	immediate	GRADE IV	CONSERVATIVE		NOT RECOVERED	
31	Perumalthai	29 M	53892	malignant growth EAC	1month	GRADE IV	CONSERVATIVE		NOT RECOVERED	
32	Rajasekar	14 M	23175	ASOM	1week	GRADE IV	SURGERY	immediate	GRADE I	
33	Velayutham	48 M	48723	Trauma	delayed	GRADE III	CONSERVATIVE	1month	GRADE I	
34	Murugan	26 M	68284	Trauma	delayed	GRADE III	CONSERVATIVE	1month	GRADE I	
35	Karthick	25 M	53637	Trauma	immediate	GRADE IV	SURGERY	3months	GRADE I	
36	Manopriyan	22 M	19432	Trauma	immediate	GRADE IV	CONSERVATIVE	2weeks	GRADE I	
37	Velladurai	35 M	28496	CSOM	10months	GRADE IV	SURGERY		NOT RECOVERED	
38	Bagavathi raja	23 M	73721	Trauma	immediate	GRADE IV	SURGERY	1month	GRADE I	
39	Kaliraj	36 M	10480	Trauma	immediate	GRADE IV	SURGERY	3months	GRADE I	
40	Cheramadurai	58 M	43218	CSOM	1week	GRADE IV	SURGERY	4months	GRADE I	